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DIAGNOSIS AND TREATMENT OF GOUT *

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Recent advances in the diagnosis and treatment of gout, together with the resurgence of interest in a variety of joint diseases, have focused attention once more upon this specific metabolic dyscrasia, which is encountered frequently by the specialist and the general practitioner. I have been interested in this disease for more than two decades and like those of you who are particularly interested in this malady, continue to be surprised by at least two items that may be documented. The first is the failure of physicians to consider gout in the differential diagnosis of each mild or severe joint affliction, particularly in the male. Secondly, the lack of appreciation by physicians of the management once the disease has been suspected. Gouty arthritis is probably the easiest joint disease to treat among the more common arthritic disorders. Surely, it is the most rewarding to the physician as well as to the patient. Hence, the diagnosis and the treatment comprise the bulk of what I have to say at this time.

DIAGNOSIS

The finding of a few white bumps in the ear of a male who has had joint distress is pathognomonic of gout. But I do not see this manifestation in a great majority of patients. Tophi in the ears usually appear after the disease has been present for a decade or more and the patient has had a goodly number of acute attacks of gouty arthritis. The disease should have been recognized and controlled before the development of subcutaneous tophi.

The observation concerning familial incidence of disease generally, in taking a complete history in a patient, usually is not rewarding. From experience we tend to overlook this item. If we fail to inquire regarding the family history in a patient suspected of having gout, however, we may miss an important clue. The incidence of gouty arthritis in families is significant and should not be overlooked. In addition, the concentration of uric acid in the serum of relatives of gouty patients, relatives who have been termed nonaffected relatives, because they have had no joint distress, likewise is significant. An illustration may be helpful:

In 1948, I was requested to provide terminal medical care for a patient in advanced renal insufficiency who had had acute attacks of gouty arthritis for almost two decades. Following the initial shock of his death, complete cooperation with

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his brothers, sons and nephews furnished revealing data. Two of the three brothers, both sons, and both nephews, six out of seven investigated, had an elevated uric acid in the serum. There was no past history of arthritis in any of them and x-rays of the feet and hands in each instance were negative. These males have been seen at intervals of from eighteen to twenty-four months and the increased concentration of uric acid in the serum has been confirmed. One of the brothers, who was 50 when he was first seen, had uric acid concentrations of 8.6, 7.4 and 7.0 mgm., respectively, between 1948 and 1953. In the fall of 1954, six years after the initial determination, his family physician reported the sudden onset of acute distress in the great toe. The diagnosis of acute gout was suspected within a matter of a few hours, because the family physician was aware of the presence of gout in the family and my interest in checking the serum levels from time to time. The patient was treated promptly with Colchicine, lost only one day from his office and has been symptom-free during the past eighteen months. He takes two Colchicine daily as a prophylactic measure. I continue to see the sons at regular intervals and the concentration of uric acid in the serum in each instance remains in the gouty range. Meanwhile, they continue to be symptom-free without any specific or general measures followed. In other words, I have considered it neither desirable nor necessary to place any of the nonaffected relatives with an elevated uric acid on any regimen of management.

The next patient is an equally good illustration. A hale, hearty, and well developed male had had acute distress in his elbow for a few days with the diagnosis of "tennis elbow". He had suffered no injury to the elbow and his family doctor made the diagnosis of olecranon bursitis by exclusion. Rather than treat the disturbance empirically, he was referred to the hospital for study. Gout was suspected but not too seriously. Inquiry was made regarding the family history of gout and it was denied emphatically. In fact, there was a little resentment that I should have suspected gout in this man. The concentration of uric acid in the serum was nearly 9 mgm. The cardinal signs of inflammation were present. I found no other evidence of joint disease and decided to try a therapeutic, as well as diagnostic, course of Colchicine. The response was gratifying and twenty-four hours later there was only slight residual in the elbow. At the time of discharge the diagnosis of gout was no longer resented, especially because an optimistic prognosis was given. The patient agreed to inquire specifically among his relatives regarding the possibility of gout having been in the family. Six months later he called me up and was pleased to report that his grandfather was known to have had gout and there was some question that his father had it in a mild form also.

At the initial examination of a patient suspected of gout, there are three ancillary items that have been helpful in providing confirmatory evidence. These are: the passage of uric acid gravel or a uric acid stone, albuminuria and hypertension respectively. We have several patients in our series who have passed a uric acid stone before the first attack of acute gouty arthritis. In two instances, stones were passed more than a decade before the joints were the center of attention. I think we have evidence now to explain this phenomenon. Uric acid is a naturally occurring substance in the healthy as well as the sick. It is one of the end-products of purine metabolism. Evidence gained during the past ten years, from isotope studies particularly, lends support to the presumption that the increased concentration of uric acid in the serum and body fluids is a result of increased production, not decreased excretion or decreased destruction by the body. The excellent studies of Stetten and his associates have demonstrated that in some gouty patients at least, the formation of uric acid proceeds through a more direct pathway than in normals. These studies have utilized glycine labeled with N-15. In our labora-

tory these observations have been confirmed. Other evidence in support of an increased formation of uric acid, an integral aspect of the pathogenesis of the disturbance, has been available for several decades. Almost from the beginning of modern clinical biochemistry, an occasional patient with gout was observed to excrete more uric acid than a control subject on a constant diet. We reported several such instances several years ago and more recently this has been confirmed by others. The increased formation of uric acid by the body is believed to be the metabolic defect that is transmitted in the family. The nonaffected relatives with an increased concentration of uric acid in the serum have the defect but it is not of a magnitude, in most instances, to produce acute attacks of gouty arthritis. This defect probably is present from birth. It is not of clinical significance until the second or third decade of life or later. The increased production of uric acid is so slight that the body is able to handle the excess for a period of years. If the defect is very slight the body presumably may handle the defect throughout life. It should be noted that we have had several patients who have had their first attack of gouty arthritis in the seventh and eighth decades of life. But let us not deviate too far from the concept of increased production of uric acid by the body. The increased production of uric acid leads to an increased concentration of uric acid in the serum and an increased excretion by the kidneys with several possible sequelae. If an adequate amount of urine is available, the excess uric acid is excreted in solution without demonstrable harm. If the quantity of urine excreted is inadequate to dissolve the uric acid that is presented to it for excretion, precipitation in the tubules or the renal pelvis is a possibility. Furthermore, deposition of uric acid in the tubules and in the pelvis certainly may precede the attacks of acute arthritis.

The second ancillary item is the presence of albuminuria. I have seen several patients who have had albuminuria one or more years before the appearance of gout. I believe that this is a result of the mechanical irritation of the uric acid crystals in the renal tubules. An elevation of blood pressure is the third ancillary item. More than 25 per cent of the patients with gout have a blood pressure that is above normal. It is essentially a benign type of hypertension and, with but one exception, does not appear to be malignant in type. Of course, the occasional patient with gout and severe renal disease may develop a pseudomalignant type of hypertension as a result of the renal insufficiency. Nor does this run the course of the garden-variety type of malignant hypertension.

The above items are part of the history and should not be overlooked. The greatest help in diagnosis, however, is obtained from the clinical characteristics of the acute attack. The sudden onset of acute distress in one of the peripheral joints of the body in a male, points to one diagnosis only. Cardinal signs of inflammation may be present with an elevation of temperature, an increase in the sedimentation rate and a leukocytosis. If the patient is not seen at the time of acute symptoms, the severity of distress may be recounted vividly by the patient. The other types of joint disease commonly encountered, acute rheumatic fever, acute rheumatoid and osteoarthritis, usually are not associated with such acute distress. Clinical observation still is useful in the practice of medicine. If the acute attack appears in the foot, the patient may resort to several devices to obtain relief. One is the operation upon the shoe which results in the "gouty shoe". If the shoe is slit or the medial aspect about the great toe is perforated, suspect gout.

The concentration of uric acid in the serum may be extremely helpful in diagnosis, but like any other laboratory procedure, should be used for confirmation of a diagnosis, not to make a diagnosis. The uric acid may be elevated in normal persons for reasons not entirely clear to me, as well as in some patients with other types of joint disease. If the patient is

suspected of having gout, and the uric acid in the serum is above 6 mgm., this is confirmatory evidence. Note that serum or plasma rather than whole blood should be used for the determination. The patient should be taking no antirheumatic drug within a forty-eight hour period of collecting blood for the determination. Each of the nonspecific agents commonly used in the treatment of joint disease, such as salicylates, phenylbutizone, and the adrenal steroids cause an increased excretion of uric acid from the body, depress the concentration in the serum and complicate the interpretation of the laboratory results.

Colchicine, on the other hand, is specific for acute gouty arthritis and has no significant antiarthritic effect in other types of joint disease. The response to Colchicine when given orally or intravenously may be extremely helpful, diagnostically as well as therapeutically. But let us return to the concentration of uric acid in the serum. In our clinic, the patient with gout has an increased concentration of uric acid in the serum above 6 mgm., irrespective of the stage of the disease, and irrespective of the time in relation to the acute attack. The only agents that modify the concentration are those that lead to an increased excretion of uric acid by the body, that is, the antirheumatic agents other than Colchicine. Colchicine has no demonstrable effect upon uric acid metabolism or the concentration of uric acid in the serum. In a large number of determinations we have found that about 95 per cent of the patients with gout show an increased concentration of uric acid above 6 mgm., except as noted above. On the other hand, not more than 4 or 5 per cent of nongouty patients have a concentration above 6 mgm. This procedure then is very helpful.

The x-ray changes in gouty arthritis have been over-emphasized. In the early years of the disease, x-ray changes are not to be expected and one may make a diagnosis with completely negative joints by x-ray. Furthermore, in rheumatoid arthritis there may be punched-out areas in some joints which lead the unsuspecting to consider gout. Rheumatoid arthritis usually lends itself to diagnosis from the clinical findings and the presence of a few punched-out areas in the bones of a patient with rheumatoid arthritis should not mislead the physician.

The extensive changes about the metatarsal-phalangeal joint of the great toe are apparent in Figure 1. Note, however, that many of the other joints are essentially untouched.

Figure 1. X-ray of the left foot of a male aged 77 who had had intermittent attacks of acute gout for more than 30 years. Moderately advanced roentgenographic changes in diverse parts of the extremities were noted. There are many osseous tophi of varied sizes, narrowing or obliteration of some joint spaces, some overgrowth of bone and decalcification.

In Figure 2 similar extensive changes are noted about some of the joints, other joints are essentially normal.

As the disease progresses more joints may be involved, (Figure 3) yet others appear essentially normal by x-ray.

In the late advanced stages, amputation

Figure 2. X-ray of the hands of patient with advanced changes of tophaceous gouty arthritis.

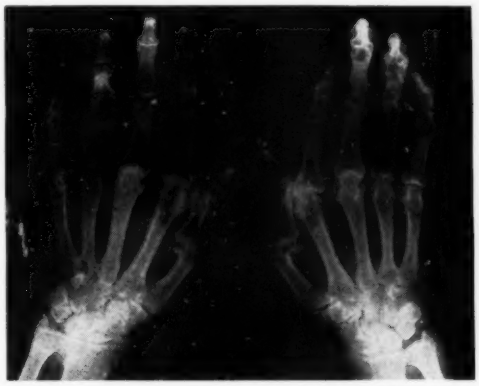

Figure 3. X-ray of the hands of a male patient aged 23 with advanced and extensive tophaceous gout who suffered involvement of a great many of the joints of the upper and lower extremities. Intermittent attacks of acute arthritis had been present for more than 8 years. Several of the joints, particularly the metacarpal-phalangeal joint of the index finger on the left, are obliterated by urate deposits. There is expansion of the cortex of the third phalanx on the right. The terminal phalanx on the right has been amputated. This was desirable for cosmetic reasons as well as because it was functionless.

sometimes is necessary as illustrated in Figure 4.

Although the hands and feet are most apt to be involved, other joints may be the site of well developed tophaceous deposits. Figure 5 is the x-ray of an ankle. Several osseous tophi are seen in the joint as well as adjacent to the articular surface.

The elbow shown in Figure 6 has a large subcutaneous tophus as well as extensive osseous tophi.

Figure 4. X-ray of the hands of a patient aged 38 with moderately advanced chronic tophaceous gout. Symptoms of intermittent acute gouty arthritis have been present for 18 years. Extensive osseous involvement is apparent. This patient has huge subcutaneous tophi overlying several of the osseous tophi.

Figure 5. X-ray of the ankles of a patient aged 50 who has advanced tophaceous gout. Symptoms of acute gout had been present intermittently for more than 20 years. Urate tophi are noted at articulations in bone structures not adjacent to articulating surfaces. The tophi have broken through the cortex and encompass soft tissues. There is destruction of the articular spaces, sclerosis and overgrowth of bone.

In Figure 7 osseous tophi are present in the anatomical neck of the humerus.

At the present time most of our x-rays of joints are taken by the magnification technique. This technique does not show up to advantage on lantern slides but upon direct inspection of the x-ray film, it is very helpful. The detail is increased by 100 per cent.

TREATMENT

The prognosis in this chronic disease is excellent. With the earlier and increased

Figure 6. X-ray of the elbow. Several large urate tophi in the olecranon process are evident. There is diffuse calcium deposition in the large subcutaneous tophus which completely surrounds the elbow.

Figure 7. X-ray of the right shoulder. Osseous tophi in the anatomical neck of the humerus are evident.

recognition, the proportion of those severely afflicted has decreased significantly. In the series of patients that I have been following during the past decade, less than 5 per cent have died prematurely or will die prematurely because of the disease or any of its complications. More than 90 per cent of those afflicted suffer surprisingly little and are able to lead normal lives if they follow a relatively simple schedule of management. We see an occasional patient who represents an exception to this generalization. Strangely enough two of the three most unhappy patients with gout whom I can recall are physicians who refuse to follow either my medical advice or the advice of any other physician. Presumably, they wish to be their own doctor and have not fared well as a result. One physician became a heroin addict because he preferred this agent to Colchicine in the treatment of his acute attacks. The other physician prefers several days of incapacity, three or four times a year because he cannot be bothered to take one or two Colchicine and Benemid tablets each day.

Over and above these unhappy examples are the vast majority of gout patients, who are cooperative and delighted to follow a relatively simple regimen in return for the opportunity to live a normal life and pursue a gainful occupation. This optimistic prognosis has been amply enhanced since 1950 when Benemid was introduced. Even before 1950 the agents that were available contributed greatly to the treatment and prevention of acute arthritis in those moderately or severely afflicted. In others, the disease appeared low-grade and did not progress in a detectable manner decade by decade. Probably in this category it made little difference what dietary or therapeutic regimen was recommended beyond that of sensible living. Mr. H. C. is an example.

This patient was referred to me nearly seven years ago at the time of his first attack of gout. He was the manager of a trucking company who could give as well as take it from his truck drivers. I think that his first attack of acute gouty arthritis caused him more misery than he had ever experienced before in his life. He felt that the devil was beginning to balance the books prematurely. With Colchicine, we were able to relieve his joint symptoms rapidly and forty-eight hours later he was ready to leave the hospital with all evidence of the acute attack gone. I recommended that he lose 25 lbs. in body weight because he was obese, a high fluid intake, alcohol in moderation only, and two Colchicine a day. The transformation that took place during the next few months surprised his friends as well as his physician. He lost the desired weight, began for the first time in 25 years to lead a sensible life, became a teetotaler, developed a profound interest in a Boy's Club and has been the epitome of rightousness for several years. With two

Colchicine a day he has not had a recurrence of his acute arthritis and for reasons not entirely clear to me, the concentration of uric acid in the serum has steadily fallen. This trend of the uric acid in the serum without the use of agents directed against uric acid metabolism is unique in my experience.

The management of acute and chronic gout is probably more satisfactory than the management of any other chronic joint disease as was noted in the introduction. There is nothing more gratifying than to find some poor soul in excruciating pain, appetite gone, having chills and fever, unable to sleep, unable to work, and thoroughly miserable day after day, and then have him undergo a remarkable transformation within twenty-four hours and be back at work within a few days. This may be accomplished with oral or intravenous Colchicine in a high percentage of instances, with Phenylbutizone in a majority of instances and with large amounts of ACTH or prednisone in a few instances. My preference for the several agents is in this order. If the acute attack has been present, untreated, or poorly treated for one or more days, recovery may be less dramatic. If the joint distress, having been present for not more than twenty-four hours, does not respond to full doses of Colchicine, the diagnosis should be questioned or some complication should be sought. If distress has been present for longer than twenty-four hours, Colchicine supplemented by Phenylbutazone or ACTH should be tried. During convalescence from acute gouty arthritis, early ambulation is to be encouraged. There are no other specific measures to be recommended for the treatment of the acute episode. General measures include, an abundance of fluids, protection of affected joints, and an ad lib diet.

The management of the intercritical period, which should encompass most of the time for all patients with gout, comprises a few drugs and a few prohibitions, in short, a simple regimen. A high fluid intake is to be stressed. This aids in the elimination of uric acid from the body, a particularly important feature when Benemid is prescribed. Weight reduction, if indicated, should be insisted upon. Substances high in purines, such as liver, kidney, and sweetbreads, should be avoided. This entails no hardship. The patients are permitted red meat once a day if they wish and alcohol in moderation. I have many instances of patients taking alcohol daily as well as Colchicine, experiencing little or no distress from gout, year by year. On the contrary, I see an occasional patient who prefers to stay away from alcohol as well as Colchicine and who gets into trouble from time to time. The amount of physical activity should be determined by factors other than the presence of gout. Hunting, fishing, and other forms of strenuous activity are permitted.

It is believed that there are only two drugs that should be used regularly in the period between attacks of acute arthritis. These are Colchicine, one of the oldest specific drugs in the pharmacopeia and Benemid, one of the newest. For patients moderately afflicted with gout, one or two tablets are prescribed daily throughout the year. Those severely afflicted take two or three tablets daily. Gastrointestinal tolerance is the determining factor for this group. Chronic intolerance to Colchicine does not develop and acute toxicity from Colchicine is confined to gastrointestinal irritability. The patients mildly afflicted, which comprise at least a fourth of the patients in my series, may take Colchicine either daily or occasionally. Colchicine reduces significantly the incidence of acute attacks regardless of the severity of the disease. Equally important, however, in prophylaxis, is the administration of Benemid.

The introduction of Benemid nearly six years ago, filled a void in the therapeutic armamentarium that had been recognized for a long time. Uricosuric agents were available before Benemid, such as salicylates and cincophen, but no single agent prior to 1950 was a practical answer to the quest for a substance that would achieve satisfactory elimination of uric acid from the body when administered in

relatively small amounts and yet be endowed with negligible toxicity. The salicylates especially have enjoyed a favorable reputation as uricosuric agents but in order to achieve a good therapeutic effect, toxic or subtoxic levels must be given daily; that is, from 4 to 8 gm. a day. Even in this quantity the effect upon uric acid may be less than that of not more than 1 or 2 gm. of Benemid. Furthermore, the possibility of escape from salicylates is real. No escape from Benemid has been recorded. Approximately 1 gm. of Benemid results in satisfactory uricosuria in most patients if taken daily. The decrease in the concentration of uric acid in the serum of 25 to 35 per cent with a concommittant increase in excretion in the urine follows.

Many patients with gout have a concentration of uric acid in the serum that ranges between 8 and 10 mgm. Under the influence of Benemid the concentration is in the normal range or near the normal range.

Mr. F. D. is an example. He was seen first in 1947. The concentration of uric acid in the serum ranged from 6 to 9.2 mgm. during the next six years. He has not lost any time from work since he was seen first; meanwhile, he has been on Colchicine daily until two years ago at which time he was placed on Benemid. In 1952 and 1953, although he was not losing any time from work, he was taking more Colchicine early in the course of acute attacks and it was decided to place him on Benemid, 1.5 gm. a day at first, and then 1.0 gm. thereafter. He has taken extra Colchicine on one day only during the past year.

During the five and one half year period that Benemid has been available, the favorable results have predominated; the untoward results have been minimal. Benemid may increase slightly the tendency to form uric acid stones. However, it should not be forgotten that approximately 15 per cent of all patients with gout have a history of calculi independent of administration of uricosuric agents. Gastrointestinal irritability appears in some on Benemid but usually can be corrected by intelligent management. An occasional patient will develop a skin rash. The vast majority of patients tolerate the drug in an excellent fashion. At this stage of our clinical investigation of the value of Benemid, it is recommended that those severely afflicted with gout remain on Benemid as well as Colchicine for an indefinite period. Those mildly afflicted are permitted considerable freedom. Patients with moderate gout may use Benemid from time to time, having been on it regularly for a period of two or three years, meanwhile maintaining one or two Colchicine tablets daily. Since the escape from Benemid is rapid and return to the gouty range follows within twenty-four hours after ingestion of the last Benemid tablet, I do not believe that a person moderately or severely afflicted should remain off of Benemid for a long period of time even though he may be clinically free from acute attacks. Since gout is a chronic disease, the disturbance of uric acid metabolism persists so long as the patient lives. The threat of an acute attack remains with the increased concentration of urates in body fluids.

And finally a word regarding surgery. We still find it necessary, in those severely afflicted, to remove large tophi or useless digits. In each instance, these patients have had the disease for long periods of time, usually twenty or more years, with extensive subcutaneous and osseous tophi and chronic sinus formation.

If a person must have a joint disease, is it not preferred to have gout, take a few pills a day, and live a normal life otherwise?

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A NEW EXPLANATION OF THE ABSENCE OF P-WAVES IN A CASE OF TAWARA-NODE RHYTHM WITH RETROGRADE CONDUCTION

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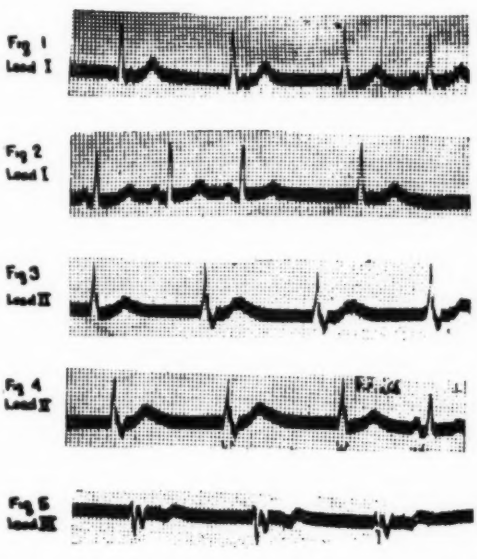
The electrocardiogram (Figures 1 to 4) shows that the explanations given for the phenomenon, mentioned above, by Paul White[1] and Charles Friedberg[2] in the

recent editions of their textbooks, do not apply to this case. Both standard works give excellent information for reading of the majority of unusual patterns of electrocardiograms. In most of the numerous smaller books about the essentials of electrocardiography this rare phenomenon is not mentioned.

The patient was a woman, 66 years of age, who had suffered for more than a year from dizziness with slight attacks of dyspnea. Other data from the history have no value for the purpose of this article.

Cases of Tawara-node rhythm with retrograde conduction are rare and the absence in these ECG's of P-waves, is still more rare.

The majority of complexes in the ECG, printed here, show a Tawara-node rhythm with retrograde conduction and with a pulse rate of 54. However, one sees in Fig. 1, 2, and 4 sometimes a positive P-wave showing the action of the sino-atrial node, which therefore is not always superseded by the action of the Tawara-node. In Fig. 1 (lead 1) the P-wave is absent in the third complex. In Fig. 3



(lead II) it is absent in the first complex. In Fig. 4 (lead II) it is absent in the third complex. In the other complexes negative P-waves follow immediately after the R-waves.

Paul White, Charles Friedberg and several others have explained the absence of the P-wave, by supposing that the P-wave is buried in the R-wave. If that explanation was right for this case, one would certainly see a much lower R-wave in Lead II, because a deep negative P would then be buried in a R-wave. This is not the case. Accurate measurements show that all R-waves of the Tawara-node rhythm do not differ in height in the same lead.

We have to look for an explanation which does not require a change in height of the R-waves of the same lead. It seems to me that the correct explanation is as follows: The part of the Tawara-node impulse which goes upward causes, in the majority of complexes, the contraction of the auricle before the sinus-node impulse reaches the auricular musculature. This auricular musculature is then refractory for the later coming sinus-node impulse. However, more rarely the impulses of the Tawara-node and sinus-node reach the auricular musculature at the same time. The electrical phenomena neutralize each other. The result is that in these cases the ECG shows no P-wave.

SUMMARY

A new explanation is given for the absence of P-waves in some complexes of an ECG that shows the Tawara-node rhythm with retrograde conduction.

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AN ANALYSIS OF 2229 AUTOPSIES WITH SPECIAL EMPHASIS ON CIRCULATORY SYSTEM AND ALLIED DEATHS

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NEW ORLEANS

This study deals with an analysis of the circulatory system, cardiac, pulmonary, renal, and related deaths autopsied

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at the Touro Infirmary from January 1, 1945, through August 31, 1955. There were 3860 deaths, with 2229 autopsies (55.15 per cent) performed.

Before the actual data collected are presented it should be stated that although Touro Infirmary operates outclinics for white and colored patients, only white patients are admitted to the wards. This is important when comparisons of these data are made with those of other hospitals where admissions are not so restricted.

Of the total deaths, 877 were circulatory in nature, 809 cardiac and 68 pulmonary. Those classified as cardiac included deaths from heart failure and associated conditions, such as uremia, cerebral hemorrhage and thrombosis, hypertension and its complications, rupture of aortic aneurysms, and the like.

The purposes of this study were to determine (1) the relative incidence of the different types of heart disease with special reference to age and sex; (2) the relative incidence of pulmonic embolic phenomena; and (3) other data on such topics as perforated myocardial infarctions, cardiac aneurysm, etc. which could be derived from this study.

Of course, it is obvious that the true incidence of any disease can be determined only by autopsy whether death occurs at

TABLE 1
HEART DISEASE AND ALLIED CAUSES OF DEATH—877 CASES

TYPES	No. of Cases	% of Group	% of Total Group	% of all Deaths	Male	Female
I. Infectious	44		05.45	01.97		
A. Rheumatic	20	45.45	02.47	00.90	10	10
B. Bacterial	8	18.18	00.82	00.36	8	0
C. Syphilitic	6	13.64	00.74	00.27	3	3
D. Myocarditis	10	22.74	01.24	00.45	3	7
Total I	44		05.75	01.97	24	20
II. Noninfectious	765		94.56	34.32	493	272
A. Hypertensive primary	499	65.31	61.76	22.39	315	184
1. Myocardial insufficiency	87	11.38	10.77	03.90	51	36
*2. Coronary sclerosis	192	25.13	23.80	08.61	134	58
3. Cerebral complications; apoplexy, thrombosis, etc.	122	16.00	15.10	05.47	70	52
4. Renal insufficiency	78	10.21	09.65	03.50	42	36
5. Dissecting and other aneurysm of the aorta	20	02.60	02.47	00.90	18	2
*B. Coronary sclerosis	154	20.15	19.06	06.91	116	38
C. Pulmonary hypertension (Cor. Pulmonale)	19	02.48	02.35	00.89	11	8
D. Congenital	54	06.94	06.56	02.38	31	23
E. Miscellaneous	31	04.06	03.83	01.35	18	13
*F. Hypertensive and arteriosclerotic by pathological diagnosis	8	01.05	00.91	00.36	2	6
Total II	765		94.56	34.32	493	272
Total I & II	809			36.30	517	292
III. Pulmonary embolus/ Infarction	68		07.76	03.05	28	40
Totals I, II, III	877			39.35	545	332
All Hypertensive and Arteriosclerotic, *11A2, IIB, & IIF	354	46.33	43.81	16.00	252	102

home, in the hospital, or elsewhere. However, in order to reach a truer relative determination, certain portions of this paper will be denoted where not only the disease was per se the actual cause of demise of the patient, but instances where definite and significant findings were present even though the patient might have died of other and perhaps unrelated causes will be discussed and related data co-ordinated and integrated. (Table 1-A):

This report is mainly divided into three groups: Infectious and noninfectious heart disease and pulmonary embolism. (See Table 1).

I. INFECTIOUS HEART DISEASE

Included in the infectious group are rheumatic, bacterial, and syphilitic heart disease and myocarditis. Of these, there were 44 deaths, or 5.75 per cent of the cardiacs and 1.97 per cent of all deaths.

Rheumatic Heart Disease: In this group of 20 were also included patients with valvulitis from acute rheumatic fever, valvular deformities resulting from repeated rheumatic infections, and probable adhesive pericarditis.[1a, b] However, as may be seen in Table 2, there were 24 more cases with significant findings of rheumatic heart disease who died from other causes, bringing the total of this type of heart disease to 44. The deaths comprised 45.45 per cent of the infectious group, 2.47 per cent of total cardiac deaths, and 0.90 per cent of all deaths.

Of the 44 cases as a whole, 21 were males and 23 females. Among the deaths, 10 males and 10 females were found. The youngest male was 7 years; youngest female 7 years; oldest male was 80 years

TABLE 1-A
HEART DISEASE & ALLIED CAUSES OF DEATH AND PATHOLOGY—956 CASES

TYPE	No. of Cases	% of Group	% of Total Group	% of all Deaths	Male	Female
I. Infectious	77					
A. Rheumatic	44	55.01	05.16	01.52	21	23
B. Bacterial	12	15.58	01.41	00.52	10	2
C. Syphilitic	11	14.29	01.29	00.49	7	4
D. Myocarditis	10	12.97	01.17	00.44	3	7
Total I	77		09.05	03.45	41	36
II. Noninfectious	775					
A. Hypertensive primary	505	65.16	59.27	22.70	320	185
1. Myocardial insuff.	87	11.23	10.21	03.90	51	36
*2. Coronary sclerosis	192	24.80	22.53	08.61	134	58
3. Cerebral complications; apoplexy, thrombosis, etc.	122	15.74	14.32	05.47	70	52
4. Renal	78	10.06	09.15	03.50	42	36
5. Dissecting and other aneurysm of the aorta	26	03.35	03.05	01.12	23	3
*B. Coronary sclerosis	154	20.00	18.07	06.91	116	38
C. Pulmonary hypertension (Cor. Pulmonale)	20	02.58	02.35	00.89	12	8
D. Congenital	57	07.78	06.69	02.56	34	23
E. Miscellaneous	31	04.00	03.64	01.39	18	13
F. Hypertensive and Arteriosclerotic	8	01.03	00.94	00.36	2	6
Total II	775		90.96	34.81	502	273
Total I & II	852			38.22	543	309
III. Pulmonary embolism	104		10.88	04.67	56	48
Total I & II & III	956			42.88	599	357
*IIA2 & IIB & IIF	354	45.68	41.55	15.84	252	102

and oldest female 75 years. The greatest number of deaths occurred in the fourth, fifth, and sixth decades. Of course, this series of cases was rather small but it was all that could be found in this study.

Bacterial Heart Disease: This group contained only 8 cases. From Table 1 and Table 3 we can easily discern that this series of autopsies contained a total of 12 cases, 8 of which died of bacterial heart disease; 7 deaths were from subacute and 1 from acute bacterial endocarditis. The youngest male was 36 years old; the oldest male 73. There were no females among the deaths, which comprised 18.18 per cent of the group; 0.82 per cent of the cardiac groups; and 0.36 per cent of all deaths. Four cases had significant findings but died of other causes.

Syphilitic Heart Disease: Cases with luetic aortitis in which death resulted comprised 6 deaths; other syphilitic pathology in the heart and/or circulatory system made up the 5 additional cases with pathology only, as seen in Table 4. The 6 deaths were composed of 3 male

TABLE 2
GROUP I. INFECTIOUS—A. RHEUMATIC

	44 Cases with Significant Findings			20 Cases died of Rheum. Heart Dis.		
Age	Male	Female	Total	Male	Female	Total
0 - 10	1	2	3	1	2	3
11 - 20		1	1		1	1
21 - 30		2	2		1	1
31 - 40	2	2	4	2	1	3
41 - 50	4	3	7	3		3
51 - 60	3	5	8	1	3	4
61 - 70	4	4	8	3	1	4
71 - 80	7	4	11		1	1
Totals	21	23	44	10	10	20
Youngest yrs	7	7		7	7	yrs
Oldest yrs	80	79		80	75	yrs

44 cases: 20 died of rheumatic heart disease.
24 showed significant findings at autopsy but died of other causes.

TABLE 3
GROUP I. INFECTIOUS—B. BACTERIAL

	12 cases with Significant Findings			8 cases died of Bact.Heart Dis.		
Age	Male	Female	Total	Male	Female	Total
31 - 40	1		1	1		1
41 - 50	2		2	1		1
51 - 60	3		3	3		3
61 - 70	2		2	2		2
71 - 80	1	1	2	1		1
81 - 90	1	1	2			
Totals	10	2	12	8	0	8
Youngest yrs	36	77		36	0	yrs
Oldest yrs	81	83		73	0	yrs

TABLE 4
GROUP I INFECTIOUS—C. SYPHILITIC HEART DISEASE

	11 cases of Significance			6 deaths of Syphilitic Ht. Dis.		
Age	Male	Female	Total	Male	Female	Total
31 - 40		2	2		2	2
41 - 50	1	1	2	1		1
51 - 60	4	1	5	2	1	3
61 - 70	2		2			
Totals	7	4	11	3	3	6
Youngest yrs	46	38		46	38	yrs
Oldest yrs	64	58		59	58	yrs

and 3 female. Of the "pathology only" 5 cases, all exhibited findings of luetic aortitis; 3 also had luetic aneurysm of the aorta; one had ectasia of the aorta. Those who died presented the following syphilitic stigmata: luetic aortic valvulitis with luetic aneurysm of the ascending aorta, aortic regurgitation and heart failure; syphilitic aortitis with rupture of luetic aneurysm of the aorta into the esophagus; clinical lues with embolic phenomena of the lungs, spleen, etc., and cardiac standstill from sudden arrhythmia and shock; luetic aortitis, aortic regurgitation and aortic insufficiency; luetic thoracic aneurysm of the aorta with rupture into the esophagus and left lower bronchus. This low relative incidence of death from syphilitic heart disease in all probability stems from the fact that these are figures of white cases only.

Myocarditis: Again referring to Table 1, there were 10 deaths in this group, representing 22.74 per cent of the group deaths, 1.24 per cent of total cardiac cases, and 0.45 per cent of all deaths. There were only 3 males and 7 females, with their distribution in age groups as seen in Table 5. The youngest case was a 4 month male and the oldest a 79 year female. One 2½ year female died of diphtheritic myocarditis; one 42 year female had Feidler's myocarditis; one 45 year female had postpneumonic myocarditis, with myocardial and renal insufficiency. One 6 year female had mural thrombosis of the right auricular appendage, the heart was enlarged, and there was evidence of questionable rheumatic endocarditis although no Aschoff bodies were found. The 4 month male showed a toxic myocarditis one week after an acute upper respiratory infection. One 28 year female had a postabortional myocarditis, plus renal failure and uremic pericarditis from *Clostridium welchii* infection. One 35 year female had postsurgical congestive failure and toxic myocarditis with a general toxemia. A 79 year female developed toxemia and heart failure on the operating table while undergoing gallbladder surgery, from a generalized, overwhelming toxemia. One 40 year male developed myocarditis with septic emboli causing coronary occlusion. This series was again small; it made up 22.74 per cent of the group, 1.24 per cent of the cardiac groups, and 0.45 per cent of all deaths.

TABLE 5
GROUP I. INFECTIOUS—D. MYOCARDITIS—
10 DEATHS

Age	Male	Female	Total
0 - 6 mo	1		1
2 - 3 yr		1	1
4 - 6 yr		1	1
21 - 30 yr		1	1
31 - 40		1	1
41 - 50	1	2	3
51 - 60	1		1
61 - 70			
71 - 80		1	1
Totals	3	7	10
Youngest yrs	4 mo	2½ yr	
Oldest yrs	55	79	

II. NONINFECTIOUS HEART DISEASE

These made up 765 cases, comprising 94.56 per cent of cardiac deaths and 34 per cent of the autopsies. This group was composed of hypertensive heart disease, coronary sclerosis without hypertension, pulmonary hypertensive disease, congenital heart disease, and heart disease associated with renal disease.

Hypertensive Heart Disease: In this group the heart weight was adopted as being over 400 grams for the males and over 350 grams for females.[2c] High blood pressure and its complications are included in this category, as are several cases of cerebral complications (See Table 8) which clinically resembled cerebrovascular accidents, with or without clinical hypertension, these latter being separately discussed further along in this paper. Also included are deaths from coronary sclerosis and thrombosis, cerebral hemorrhage and thrombosis, encephalomalacia, uremia, and dissecting along with other types of aneurysm of the aorta. (Table 1). There were 499 cases in this group, comprising 65.31 per cent of Group II, 61.76 per cent of the cardiac groups, and 22.39 per cent of all deaths. Males accounted for 315 cases and females 184 cases, a ratio of 1.7:1.

Myocardial Insufficiency: Table 6 shows the 87 cases found in this category; the data are self-explanatory. Of interest are the case of acromegaly in a male, and one case, a colored female aged 50 years, included in this group who died in the clinic.

TABLE 6
GROUP II. NONINFECTIOUS—A. HYPERTENSIVE PRIMARY—1. MYOCARDIAL INSUFFICIENCY—87 DEATHS

Age	Male	Female	Total
31 - 40	1	1	2
41 - 50	6	3*	9
51 - 60	17	7	24
61 - 70	18	9	27
71 - 80	8	12	20
81 - 90	1	4	5
Totals	51	36	87
Youngest yrs	39	31	yrs
Oldest yrs	86	88	yrs

* 1 colored female in this group, aged 50, died in the clinic.

Coronary Sclerosis: Table 7 describes the 192 cases, 134 male and 59 female (2.7:1) in this category. The greatest majority occurred in the fifth, sixth, and seventh decades of life. The youngest male death was 36 years; the youngest female 34 years. The oldest male and female lived to the respective ripe old ages of 91 and 88 years.

Cerebral Complications: 122 cases who died are described in part A of Table 8; the 10 cases of congenital or other cerebral aneurysms included in this group are delineated in part B of the same table. Altogether, there were 70 males and 52 females (1.35:1); there were 5 males and 5 females in the subgroup. Again, the greatest incidence of death occurred in the span between the fifth and seventh decades.

Renal Insufficiency: In Table 9 are depicted the deaths from this cause. There were 42 male and 36 female deaths. The youngest male was 3½ years; the youngest female 2 years; the oldest male and female were 75 and 88 years, respectively. The deaths occurred mostly between the second and sixth decades.

Dissecting Aneurysm of Aorta: The study disclosed 26 cases with dissecting or other aneurysm of the aorta, the deaths being divided into 18 males and 2 females,

TABLE 7
GROUP II. NONINFECTIOUS—A. HYPERTENSIVE PRIMARY—2. CORONARY SCLEROSIS—192 DEATHS

Age	Male	Female	Total
30 - 40	2	1	3
41 - 50	16	3	19
51 - 60	39	9	48
61 - 70	35	19	54
71 - 80	33	19	52
81 - 90	4	6	10
91 - 100	1		1
Totals*	130	57	187
	4*	1*	5*
	134	58	192
Youngest age	36	34	yrs
Oldest age	91	88	yrs

* 4 males and 1 female ages not given, but estimated at autopsy.

TABLE 8
GROUP II. NONINFECTIOUS—A. HYPERTENSIVE PRIMARY—3. CEREBRAL COMPLICATIONS—122 DEATHS

	A. All 122 cases			B. Congenital/Other Cerebral Aneurysms —(10 cases included)		
Age	Male	Female	Total	Male	Female	Total
21 - 30	3	1	4	1	2	3
31 - 40	3	2	5	1		1
41 - 50	12	3	15	2	1	3
51 - 60	15	9	24		2	2
61 - 70	16	13	29	1*		1
71 - 80	16	16	32			
81 - 90	3	7	10			
91 - 100	2	1	3			
Totals	70	52	122†	5	5	10
Youngest yrs	24	24		24	24	yrs
Oldest yrs	93	91		62	53	yrs

* 62 year white male died of ruptured saccular dilatation/aneurysm of cerebral artery—not hypertensive.
† 1 case each male and female, no age given; age estimated at postmortem.

TABLE 9
GROUP II. NONINFECTIOUS—A. HYPERTENSIVE PRIMARY
4. RENAL INSUFFICIENCY—78 DEATHS

Age	Male	Female	Total
0 - 10	3	3	6
11 - 20	2		2
21 - 30	5	3	8
31 - 40	8	5	13
41 - 50	8	2	10
51 - 60	7	9	16
61 - 70	5	7	12
71 - 80	4	4	8
81 - 90		3	3
Totals	42	36	78
Youngest	3½	2	yrs
Oldest	75	88	yrs

a total of 20 and a ratio of 9:1 (Table 10). The 6 cases who died of other causes but showed the aneurysms are also tabulated. Interesting is the recorded fact that only 4 cases were due to lues, all were males. Also, a 71 year old male who died of a large dissecting aneurysm of the thoracic aorta had positive serology and luetic findings, together with generalized arteriosclerosis; he was diagnosed by the pathologist as having an arteriosclerotic type of aneurysm.

Coronary Sclerosis without Hypertension: These cases accounted for 154 deaths; 116 were males and 38 females, a significant difference of 3:1. In this group, there was no history of hypertension and the hearts weighed less than 400 grams for the male and 350 grams for the female. This group made up 20.15 per cent of Group II, 19.06 per cent of the entire cardiac group, and 6.91 per cent of the autopsies. Pathologically, these cases presented myocardial infarcts with or without coronary occlusion; others showed partial to total coronary artery occlusion and no myocardial infarction. However, many of these hearts had suffered repeated occlusions and/or myocardial infarcts, and were very fibrotic (Table 11). The

TABLE 11
GROUP II. NONINFECTIOUS
B. CORONARY SCLEROSIS—154 DEATHS

Age	Male	Female	Total
31 - 40	3	1	4
41 - 50	16		16
51 - 60	43	13	56
61 - 70	26	12	38
71 - 80	23	11	34
81 - 90	4	1	5
91 - 100	1		1
Totals	116	38	154
Youngest	37	33	yrs
Oldest	92	82	yrs

greatest incidences were in the fifth, sixth and seventh decades. The oldest male was 92 years and the female 82 years; the youngest male and female were 37 and 33 years, respectively.

Pulmonary Hypertension: There were only 20 cases, 19 dying of this condition, 1 of other causes. These all had in common findings in the lungs which would cause pulmonary hypertension, hypertrophy of the right cardiac ventricle, no cardiac valvular deformities, and death resulting from cardiac failure (cor pulmonale). There were 2 acute cases, both female; one 45 year old female developed right heart failure and died on the second postoperative day; the other was a 55 year old female with extensive pulmonary

TABLE 10
GROUP II. NONINFECTIOUS—A. HYPERTENSIVE PRIMARY
5. DISSECTING/OTHER ANEURYSM OF AORTA—26 CASES/20 DEATHS

	All Cases — 26			Died — 20			Pathology at Autopsy		
Age	Male	Female	Total	Male	Female	Total	Male	Female	Total
41 - 50	2		2	2		2			
51 - 60	2	0	2	1		1	1		1
61 - 70	11	1	12	10	1	11	1		1
71 - 80	8	2	10	5	1	6	3	1	4
Totals	*23	3	26	*18	2	20	5	1	6

*1 colored male died suddenly in the clinic.
13 ruptured/dissecting aneurysms of thoracic/abdominal aorta
7 ruptured saccular aneurysms of thoracic/abdominal aorta

20 deaths

fibrosis who went into acute right heart failure and died.

There were 11 males and 8 females who died (Table 12). Table 1 gives the relative

TABLE 12
GROUP II. NONINFECTIOUS
C. PULMONARY HYPERTENSION (COR PULMONALE)
20 CASES—19 DEATHS

Age	Male	Female	Total
41 - 50		1	1
51 - 60	3	4	7
61 - 70	*5	1	6
71 - 80	2	2	4
81 - 90	2		2
Totals	12	8	20
Died	11	8	19
Youngest	51	45	yrs
Oldest	88	80	yrs

* 1 case, 68 year old male, died of other causes.
Acute: 2 females, ages 45 and 55 years.
Chronic: All other cases tabulated.

percentages. Pulmonary tuberculosis, pulmonary fibrosis and emphysema were the etiological factors in these deaths. There were no deaths following pulmonary emboli or pulmonary artery arteriosclerosis in this group.

Congenital Heart Disease: In this category were 57 cases, 54 deaths. No prematures were included, nor were newborns with the findings of patent foramen ovale and patent ductus arteriosus only, as this combination is often a normal physical finding, especially in premature infants. As in Table 1 and Table 13, there were 31

TABLE 13
GROUP II. NONINFECTIOUS
D. CONGENITAL HEART DISEASE
57 CASES—54 DEATHS

Age at death	Male	Female	Total
Newborn - 1 day	3	4	7
Over 1 day - 1 mo	10	5	15
Over 1 mo - 6 mo	9	9	18
Over 6 mo - 1 yr	6	1	7
Over 1 yr - 5 yr		4	4
Over 5 yr - 10 yr			
11 - 20 yr			
21 - 30	1		1
31 - 40			
41 - 50	1		1
51 - 60	1		1
Totals	31	23	54
Youngest	4 hr	6 hr	
Oldest	52yr	4½ yr	

3 males, aged less than 1 day, 50 years, 75 years, died of other causes but had significant congenital heart disease findings.

males and 23 females who died. Not included were 3 males, aged less than 1 day, 50 years, and 75 years respectively, who died of other causes while exhibiting findings of congenital heart disease. This group made up 6.56 per cent of cardiac deaths and 2.38 per cent of all deaths. The oldest male and female were 52 years and 4½ years respectively. The youngest male and female were 4 and 6 hours. The types of heart defects were by far found in combinations and are tabulated in order of frequency as seen in Table 14.

TABLE 14
CONGENITAL HEART DISEASE—
INCIDENCE OF DEFECTS FOUND

(a)	Inter-ventricular septal defects	21 times
(b)	Hyptertrophy of heart, auricles, and ventricles	20 "
(c)	Patent ductus arteriosus	18 "
(d)	Deformity, defect of great vessels, and anomalies	17 "
(e)	Patent foramen ovale	15 "
(f)	Defects of valves or cusps	15 "
(g)	Inter-auricular septal defects	9 "
(h)	Transposition of great vessels	9 "
(i)	Tetralogy of Fallot	6 "
(j)	Coarctation of the aorta	5 "
(k)	Two or three-chambered heart	4 "
(l)	Truncus arteriosus	3 "
(m)	Endocardial fibroelastosis	3 "
(n)	Lutembacher's syndrome	2 "
(o)	Eisenmenger's complex	2 "
(p)	Patent ductus venosus	1 "
(q)	Dextrocardia alone	1 "
(r)	Fibromyxoma of auricle	1 "

Miscellaneous: As seen by Table 1, there were 18 males and 13 females * in this aggregation, all presenting difficulty in placement in any of the other groups or components. In the order of frequency, Table 15 lists the cause of death in these cases.

Hypertensive and Arteriosclerotic: As can be seen by Table 1, these comprised only 8 cases. They were the only true combinations of such types of heart disease in the entire autopsy series, and the deaths could not be attributed to any but the combination of causes, pathologically demonstrated. As seen by Table 16, there

* Actually there were a total of 33 cases in this group, found on correction of the tabulations; 20 males and 12 females.

TABLE 15
MISCELLANEOUS—33 CASES—
CAUSE OF DEATH FITTING NO PREVIOUS CLASSIFICATION

(a) Cardiac arrest—5 surgical; 2 medical	7 cases
(b) Thrombosis of abdominal aorta—2 post-surgical; 2 from peripheral venous thrombosis	4 cases
(c) Congestive failure, hypertrophy and dilatation of the heart, cause undetermined	4 cases
(d) Multiple emboli and infarcts of the lungs, kidneys, spleen, and other viscera	4 cases
(e) Brain hemorrhage from thrombocytopenic purpura	2 cases
(f) Brain hemorrhage from leukemia	1 case
(g) Congestive heart failure from mural thrombi, metastatic carcinomatous nodules in the heart	1 case
(h) Same as "g" above, but with metastatic Kaposi's sarcoma	1 case
(i) Chronic heart failure from arteriovenous anastomosis of the wrist, congenital	1 case
(j) Hypertrophied heart from unilateral renal disease	1 case
(k) Congestive heart disease from beriberi associated with chronic alcoholism	1 case
(l) Combined cerebral infarcts and congestive heart failure from severe amebiasis	1 case
(m) Eroded arch of aorta	1 case
(n) Aortic calcified stenosis with coronary sclerosis	1 case
(o) Massive abdominal aortic aneurysm; uremia from septicemia	1 case
(p) Air embolus of heart, lungs, brain from spinal puncture	1 case
(q) Rupture of aorta into mediastinum	1 case
	33 cases

TABLE 16
GROUP II. NONINFECTIOUS
F. HYPERTENSIVE AND ARTERIOSCLEROTIC
8 CASES, ALL DEATHS; DIAGNOSED BY PATHOLOGIST AT AUTOPSY IN THIS DOUBLE CATEGORY, AS EXHIBITING FINDINGS CHARACTERISTIC OF BOTH GROUPINGS

Age	Male	Female	Total
51 - 60		2	2
61 - 70		2	2
71 - 80	2	1	3
81 - 90		1	1
Totals	2	6	8

were 2 males and 6 females.

GROUP III. PULMONARY EMBOLUS AND INFARCTION

These cases numbered 104 total and 68 deaths. The series would have been considerably larger in total cases, including deaths and pathological findings only (where patients died of other causes); the reason was simply that small lung embolic phenomena were purposely omitted from this report. Only those cases presenting definite, unmistakable and important findings or which died from this condition were included. Looking at Table 1 again, and then at Table 17, we can see that there were in all 56 males and 48 females; in the 68 deaths, 28 were males and 40 females, a significant difference of 7:10.

When divided medically and surgically, the overall group contained 63 cases (37 males and 26 females) in the former category and 41 cases (19 males and 22 females) in the latter. Divided similarly, the death group were comprised of 36 medical deaths (16 male and 20 female)

TABLE 17
GROUP III. PULMONARY EMBOLUS/INFARCTION
104 CASES—68 DEATHS

	Entire Group—104 Cases									Cause of Death—68 cases					
	By Sex			Medical 63			Surgical 41			Medical 36			Surgical 32		
Age	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
Newborn - 1 wk	1		1	1		1				1		1			
5 - 6 yr		1	1		1	1				0	1	1			
21 - 30 yr		2	2		2	2					1	1			
31 - 40	1	3	4		2	2	1	1	2		1	1		1	1
41 - 50	2	6	8	1	1	2	1	5	6	1	1	2	1	4	5
51 - 60	13	10	23	9	3	12	4	7	11	2	2	4	2	6	8
61 - 70	30	17	47	22	9	31	8	8	16	10	9	19	4	8	12
71 - 80	8	7	15	3	6	9	5		5	1	4	5	5		5
81 - 90	1	1	2	1	1	2				1	1	2			
91 - 100		1	1		1	1		1	1					1	1
Totals	56	48	104	37	26	63	19	22	41	16	20	36	12	20	32

and 32 surgical (12 male and 20 female) deaths. The greatest numbers of cases in all groups occurred in the fifth and sixth decades of life.

The occurrence of pulmonary embolic phenomena seems to have been rarely, if ever, diagnosed ante mortem. This same observation was made in papers written by several observers, all of whom admit and emphasize the extreme difficulty of diagnosis, especially when in the presence or co-existence of other complications.[4a, b, c] The clinical picture did not seem to be in accord with the older teachings, such as the patient "grasping the chest and yelling in anguish", then gasping and expiring. Instead, concurring with other investigators,[4b] the picture was so varied and at times so practically symptomless as to defy diagnosis. Actually, if not found to be sudden and immediate death upon the operating table or shortly thereafter, or after a period of several weeks or months of confinement in bed for illness or a fractured bone, the signs ran more towards these examples: A 50 year old female sitting in bed suddenly collapsed and expired eight days after perineorraphy, showing at autopsy massive bilateral pulmonary embolus from phlebothrombosis of the right femoral vein; immediate death on the operating table at any age; pulmonary infarctions from venous thromboses in a 55 year old female while lying in bed; sudden death in bed in a 57 year old female eight days after laparotomy; very quiet death from numerous pulmonary emboli in a 63 year old male with progressive peripheral thrombosis; sudden drop of blood pressure, weak pulse, vomiting, and cyanosis in a 51 year old male three days after surgery, who also had clinical lues and diabetes. An 86 year old male suffered massive pulmonary embolism and infarction complicated by a fairly recent myocardial infarction and arteriosclerotic cardio-renal disease with few complaints; a 67 year male had massive pulmonary embolism with some chest pain, had an old myocardial infarction and a normal EKG on history and physical examination, complained of newly felt chest pressure also; a 6 year old female developed massive pulmonary embolism with acute cor pulmonale and died with little complaint.

In summation, the patients died very suddenly or else had comparatively little complaints for the clinician to investigate, whether there were other complications present or not. In this series, operations, bed rest for fractures in elderly patients, thrombo-embolic phenomena in other organs, confining diseases, development of "cough or cold" while in bed after surgery (duration of several hours to two weeks, averaging five to eleven days postoperatively), and sudden death in the absence of heart and lung disease or chest complaints should make one very suspicious of pulmonary embolic phenomena. Of necessity, the usual suspicions of cardiac or allied developments should not be superseded or neglected. Of the cases tabulated in this study, only four times was there a notation on a patient's chart where pulmonary embolism was suspected while the patient was still alive; once there was a positive diagnosis of this condition four hours after surgery, several hours before death.

The roentgenologists cannot make a diagnosis of pulmonary embolism per se, so they maintain; a suspected "pneumonia spot" seems to be as close as they can arrive. Perhaps a suspicious, alert attitude of the physician, together with a close co-operation between the surgeon, the cardiologist, the attending physician, and the roentgenologist and the use of anticoagulants and antibiotics by such a team as recommended by some writers[4a, b] may help the patient, if at all possible. Nevertheless, here we are confronted by a medical challenge which is not to be disregarded; the existence of many more instances of this phenomenon than heretofore suspected must be seriously taken account of. Pulmonary embolism and infarction is indeed something to be reckoned with both from medical and surgical standpoints.

ADDITIONAL DATA FROM THIS STUDY

There are some highly informative and

enlightening as well as interesting data worthy of separate discussion which will be additionally taken up now.

1. *Diabetes Mellitus*: The number of cases with this entity were not tabulated; they were exceedingly highly represented. Nearly every type of cause of death in this study had a fairly large proportion of cases complicated by diabetes mellitus. Perhaps the large number of Jewish patients had relationship to the findings. In any event, the figures were glaring.

2. *Syphilis*: This disease was conspicuous by its relative scarcity. Again, the fact that this hospital admits only white patients is probably of direct bearing.

3. *Cardiac Aneurysm*: Although not a cause of death, there were 7 cases in all. Six of the 7 occurred in the group of arteriosclerotic hearts; 5 of these followed myocardial infarction or infarctions, and were present in varying sizes in the left ventricular wall (4 cases) and in the tip of the left ventricle (1 case). The remaining 2 cases were due to hypertension primary deaths and consisted of interventricular septal aneurysms into the right ventricles, from hypertensive hypertrophy and dilatation of the left ventricles with their increased left intraventricular pressures (Burnside syndrome). One male lived to be 76 and a female to 73 years; these had several episodes of repeated myocardial infarctions, finally dying of the same recurrence.

4. *Cardiac Arrest*: These were included in the "Miscellaneous" category but are described here. There were 7 cases in all, 5 dying on the operating table in spite of all resuscitative efforts; the other 2 were medical. Of these, one resulted from auricular fibrillation and sudden standstill of the heart from undetermined cause in a 34 year old female; the second developed cardiac arrhythmia and standstill from clinical lues and multiple embolism of the lungs, spleen, and other viscera, and was a 38 year old female. The youngest case was a 5 month male undergoing surgery for persistent hypertrophy of the thymus, who died postoperatively.

5. *Cardiac Rupture*: These 21 cases present another diagnostic and therapeutic problem. Seven cases were found among the arteriosclerotic myocardial infarctions and 14 cases among the hypertensive coronary group following infarctions. They accounted for 0.94 per cent of all deaths, 2.6 per cent of cardiac deaths, and 6.0 per cent of the hypertensive/arteriosclerotic/coronary thrombosis / myocardial infarction group (the last line of Table 1). Seven occurred in the five year period, 1945-1950; 14 after 1950.

Anticoagulants were used in only 5 of these cases. The electrocardiograms revealed only tracings compatible with myocardial infarctions. None of these was diagnosed ante mortem. Of the majority, they lived from a few minutes to six to eight hours after admission, although one male survived eleven days and died suddenly, with pathologic evidence of a myocardial tear which was several days old. Three cases arrived DOA in the ambulance; 2 died in the admitting room.[3a, b, c] One finding common to all cases who survived long enough to be admitted to their rooms was a definite downhill course without any response to antishock, oxygen, etc. therapy. These 21 cases present another diagnostic and therapeutic challenge to medicine.

6. There were 4 cases of cryptococcosis (torulosis) among the 2229 autopsies. These exhibited symptoms and signs of a meningitis, which did not respond to any treatment. They were proven by smear and culture. None were diagnosed until they reached necropsy.

COMPARISON WITH OTHER AUTOPSY STUDIES

By examining Table 18, comparison with three other autopsy series[2a, b, c] all much larger than the present study, can be easily evaluated. Clawson, Holoubek, and Wafer all show a lower incidence of combined hypertensive and arteriosclerotic heart disease, and higher incidences of rheumatic, bacterial, and syphilitic heart disease by rather significant differences. Holoubek presents results of his work from data of records from Charity Hospital at New Orleans with its high colored admissions. His study reveals a much

TABLE 18
COMPARISON WITH OTHER AUTOPSY SERIES

Etiologic Types of Heart Disease	Present Series 2,229 autopsies 809 Cardiac deaths 1945 thru 8/31/55 New Orleans Touro Infirmary	Wafer 13,015 autopsies 3,607 Cardiac deaths 1941 thru 1947 Los Angeles	Holoubek 8,313 autopsies 1,045 Cardiac deaths 1935 thru 1940 New Orleans	Clawson 30,265 autopsies 4,678 Cardiac deaths 1910 thru 1938 Minneapolis
1. Hypertensive	61.76%	70.8%	40.5%	55.5%
2. Arteriosclerotic	21.20%	4.3%	20.7%	6.2%
3. Combined 1 & 2	82.96%	75.1%	61.2%	61.7%
4. Rheumatic	2.47%	8.8%	11.1%	18.6%
5. Bacterial	0.82%	5.4%	2.1%	11.0%
6. Syphilitic	0.74%	3.1%	18.2%	7.0%
7. Congenital	6.56%	1.9%	2.2%	——

lower hypertensive and arteriosclerotic heart disease incidence, but much higher (24.73 times) syphilitic heart disease. Wafer and Houloubek are much lower in their congenital heart disease categories. Clawson's series is much higher in the rheumatic heart disease grouping probably due to the higher prevalence of rheumatic fever in the Minnesota and northern U.S.A. sector, and/or due to the lack of sulfonamides and other antibiotic drugs which became available so much later after his studies were made.

SUMMARY

A total of 2229 autopsies performed on 3860 deaths (55.15 per cent) at the Touro Infirmary were studied and the relative incidence of the different types of heart disease was determined and reported. The age and sex incidence of these several types was also tabulated.

Pulmonary embolic phenomena were categoried and tabulated as to age, sex, medical and surgical deaths, and pathological findings cases. The problems of diagnosis and possible treatment for a rather highly important but undiagnosed group of cases were emphasized and recognized as a medical challenge.

Interesting and important cases such as hearts ruptured after myocardial infarction, aneurysm of the heart wall, and cardiac arrest, etc. were described and discussed briefly. The medical challenge presented by 21 cases of death from ante mortem undiagnosed ruptured hearts was recognized and emphasized.

It is sincerely hoped that medical science will evolve suitable and successful replies to the medical challenges which have been presented. It is felt that the figures and tabulations listed in this study might have some medical significance. It remains for studies of this type in the foreseeable future to demonstrate gratifying improvements and decreases, in the present dearth, of heart disease and related killers.

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ACQUIRED AFIBRINOGENEMIA WITH COUVELAIRE UTERUS FOLLOWING CORTISONE THERAPY AND PITOCIN INDUCTION FOR AN Rh ISOSENSITIZATION

ARMAND G. McHENRY, JR., M. D.*
MONROE

Acquired afibrinogenemia in pregnancy has been described in the following pathologic states: abruptio placenta,[5] amniotic fluid embolism,[7] prolonged death of a fetus in utero with[9] or without Rh isosensitization,[2] fulminating eclampsia,[1] abortion,[2] and retained placenta.[3]

This case is of particular interest in that the patient had an Rh incompatibility and was placed on cortisone therapy as a prophylactic measure. An elective pitocin induction was begun and the fetal heart tones disappeared when she developed evidence of toxemia and abruptio placenta. After vaginal delivery of an erythroblastotic stillborn, an uncontrollable postpartal hemorrhage resulted. A postdelivery hysterectomy was performed and a Couvelaire uterus was noted. An acquired hemorrhagic diathesis due to afibrinogenemia was encountered.

CASE HISTORY

Present Illness: The patient, a 28 year old white female, was admitted at approximately 37½ weeks gestation for an elective induction of labor, the reason being that she had had two living children with erythroblastosis fetalis and two erythroblastotic stillborn infants.

Past Obstetrical History: Briefly, she had a normal delivery with the first pregnancy. The second pregnancy terminated in the birth of a viable female infant which developed jaundice and anemia. Multiple blood transfusions were given. The third baby was a stillborn. The mother was placed on 100 mg of cortisone daily at the 32nd week of the fourth pregnancy. She developed a psychosis with a marked disturbance in electrolyte balance during the 37th week of gestation. The patient received a pitocin induction which resulted in the birth of a viable female infant. The Coombs' test was positive and an exchange transfusion was successfully carried out. With the fifth pregnancy, she was advised to take cortisone therapy beginning at the 28th week of gestation but refused because of the sequelae that had occurred previously. This pregnancy terminated in an erythroblastotic stillborn with hydrops fetalis.

Prenatal Course: Patient had blocking antibodies, 1 to 64, and negative regular antibodies during the entire pregnancy. She was placed on cortisone 75 mg. daily at 28 weeks' gestation. This was increased to 100 mg. daily at 30 weeks. She also received weekly injections of progesterone 50 mg. She was placed on multivitamins as well as vitamin E orally. The weight gain was normal and there was no evidence of toxemia. Blood pressure and urine analyses were normal.

Course in Hospital: Patient entered the hospital May 5, 1954. An infusion of 1000 cc. 5 per cent dextrose in distilled water with 10 minims of pitocin was started at the rate of 15 to 18 gtts. per minute. At 12 noon she was checked and the cervix was found to be 2 cm. dilated and 10 to 15 per cent effaced. The membranes had been previously stripped but were not ruptured because of a floating head. At about 3 P.M. she developed a boardlike uterus. At this time the fetal heart tones could not be elicited. The blood pressure was 120/80. She was typed and matched. The pitocin was discontinued. The uterus was marked to see if any enlargement took place. Subsequently, she was found to be 4 cm. dilated and the membrances were ruptured artificially. Prior to this about 75 to 100 cc. of bright red blood had been lost. The blood pressure was 138/90 and the pulse was 72. Heart tones were still absent. The patient received intramusular injections of pitocin of one-half a minim at intervals of fifteen to thirty minutes from 7:00 P.M. until 11:00 P.M. During this time the blood pressure rose from 130/90 to 150/90. The pulse was regular.

She received 500 cc. of compatible O negative blood slowly. The transfusion was running at the time of delivery at 11:00 P.M. She was delivered of a stillborn infant which was not jaundiced. The amniotic fluid was blood tinged and icteric. The baby had a large liver and spleen filling its entire abdomen. Following this the placenta was delivered uneventfully. It was a very large hydropic placenta with a large central blood clot. At this time the patient began bleeding excessively. She lost about 1000 cc. of blood in a matter of a few minutes. She was not bleeding from the site of a small episiotomy nor from the cervix. The uterus was slightly enlarged and boggy, and the blood was literally pouring out. She received two bottles of Dextran and 1000 cc. of plasma. The patient continued to ooze through a uterine pack and was in a state of shock. She received blood in one arm and four ampoules of Levophed by means of a cut down, and also, 20 cc. of calcium gluconate, large amounts of vitamin K and 40 cc. of adrenal cortical extract. The blood absolutely would not clot. Fresh blood, 5000 cc. was rapidly administered. Fibrinogen was being flown by airplane from New Orleans. At 5:00 A.M. the watery sanguinous fluid had increased. Her general condition was worse. Under

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practically no anesthesia at all a laparotomy was performed. A classical Couvelaire uterus was encountered with hemorrhage out into the broad ligaments. A rapid subtotal hysterectomy was performed. Four grams of fibrinogen were administered. After completion of surgery, she continued to ooze through a vaginal pack. Presumably the bleeding was from the cervix and vaginal vault. A balloon was inserted into the rectum and slight traction applied. By 10:00 A.M. she stopped bleeding and was returned to her room at 2:00 P.M.

Course in Hospital After Surgery: Levophed was discontinued the following day. The balloon in the rectum was removed as was the vaginal pack. Oxygen was discontinued on the second day and suction removed the third day. Fluids were not forced and the urinary output was excellent. She was not morbid. On the seventh day she started passing feces per vagina. She was discharged on the tenth postoperative day.

Laboratory Data: May 6, 1954 R.B.C. 4.45 million W.B.C. 15,000 Differential normal. Urine negative. May 6, 1954, Fibrinogen 740 mg. May 7, 1954, Fibrinogen 240 mg. Bleeding, clotting and prothrombin time normal. Platelet count 200,000. R.B.C. 5 million. Hematocrit 48.

Follow-Up: Aug. 1, 1954, B.M.R. -5, 17 ketosteroids normal. P.B.I. 6 gamma. Repair of rectovaginal fistulae performed. Jan. 1, 1955, fistulae repair successful. B.P. 100/60. Patient shows no evidence of Sheehan's syndrome or pituitary necrosis.

DISCUSSION

1. *Cortisone Therapy.*—To my knowledge this is the only reported case of a pregnant patient developing an abruptio placenta, afibrinogenemia, and a Couvelaire uterus while on this therapy. Whether the cortisone was the modus operandi in producing the toxemia while receiving the pitocin induction is open to question. She showed evidence of poor wound healing which may be ascribable to this medication.

The past obstetrical history is significant and warrants a brief discussion. The first pregnancy and delivery were uneventful. Her second baby became icteric several days postdelivery and received several transfusions. The third pregnancy terminated in the delivery of a stillborn infant. An autopsy revealed typical findings of erythroblastosis. The patient was placed on cortisone 100 mg. daily beginning the 32nd week of the fourth pregnancy. She developed a psychosis and a marked disturbance in her electrolyte balance during the 37th week. She received a pitocin induction which "took" and she was delivered of a viable infant. The Coombs' test was positive. An exchange transfusion was done and the infant survived with no sequelae. Cortisone was suggested about the 30th week of the fifth pregnancy but was refused because of the previous difficulties encountered. The heart tones disappeared while she was in labor and a hydropic erythroblastotic infant was delivered. The patient agreed to take cortisone with her sixth pregnancy which terminated in a toxic abruptio placenta and a stillborn infant. An autopsy revealed erythroblastosis fetalis. The ramifications of this sordid obstetrical history could have been far reaching in that she lost a baby due to an Rh isosensitization, had a live baby while on cortisone and then lost another. Had this last infant survived I believe it would have been significant.

My experience with cortisone not only in this case but in others has been discouraging when given for Rh isosensitizations. The viable infants that I have obtained in obstetrical patients who had lost previous babies due to erythroblastosis all received exchange transfusions and had clinical evidence of erythroblastosis.

2. *Pitocin Induction*: Nowadays pitocin inductions are considered to be rather innocuous. Yet this patient had an initial blood pressure of 100/60, negative urine analysis and good fetal heart tone. She developed a tetanic uterus with severe abdominal pain, elevation of blood pressure, cessation of fetal heart tones and vaginal bleeding. In other words, she developed typical signs and symptoms of a toxic abruptio while receiving the intravenous pitocin drip.

Cases of afibrinogenemia due to amniotic fluid embolism have been described where tumultuous labors have been a factor.[4] Pitocin could contribute abnormalities of the clotting mechanism by forcing thromboplastin out into the maternal circulation from the placental site. It would

be interesting to know how many cases of afibrinogenemia received pitocin with a hard or rapid labor.

3. *Blood Coagulation Factors.* — The mechanism of the depletion of fibrinogen apparently is as follows: Thromboplastin from the placenta or decidua escapes into the maternal circulation.[6] Thus prothrombin is converted into thrombin which converts fibrinogen to fibrin with multiple fibrin emboli produced.[8] Following this, intractable hemorrhage ensues due to a fibrinogen deficiency.

It is significant that the fibrinogen level was increased at the time of the symptoms of abruptio beginning approximately ten hours prior to delivery. Do fibrinogen levels always rise with, or herald, impending toxic abruptio with hemorrhage? Would it be possible to be forewarned of afibrinogenemia by an initial hyperfibrinogenemia?

This patient manifested evidence of a marked fibrinogen deficiency for an interval of eight hours as evidenced by failure of the venous blood to clot.

Unfortunately adequate blood studies were not made in this case. An improvement was noted when the fibrinogen was finally obtained.

4. *Plan of Management.* — In retrospect, this patient should have been sectioned when she manifested evidence of a toxic abruptio because it appears certain that the Couvelaire uterus was also present. However, she was a multipara with a dead baby and was 4 cm. dilated and was not in shock so the membranes were ruptured and she was given I.M. pitocin. Should the hysterectomy have been done after the cervix was inspected and the uterus packed unsuccessfully? Fresh blood was being given to her as rapidly as possible and she was kept alive on levophed. Fibrinogen was not available and she had the obvious afibrinogenemia.

CONCLUSION

A case of acquired afibrinogenemia with an abruptio placenta, Couvelaire uterus that had been on cortisone therapy for an Rh isosensitization was presented. An elective pitocin induction was done prior to the abruptio.

Numerous intriguing factors were provoked by this case and a severe defect in the clotting mechanism was encountered.

In conclusion, all hospitals where obstetrics is practiced should have blood banks with adequate amounts of all types of blood. Levophed should be in the delivery rooms with large needles and glucose. Fibrinogen is now available and should be kept in all hospitals in adequate amounts.

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BEQUEST OF THE BODY AT DEATH

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NEW ORLEANS

Members of the medical profession in Louisiana are in position to aid in a situation that seriously affects medical and dental education in the State. The study of anatomy is a basic essential in education for these professions, and it can be pursued only by dissection of the human body. Although the law[1] provides that unclaimed bodies be placed at the disposal of the three Louisiana schools that offer anatomical instruction, the supply has

* Secretary-Treasurer, Louisiana State Anatomical Board, and Professor of Anatomy, School of Medicine, Tulane University, New Orleans.

been inadequate. Our schools are not alone in this, for the shortage of anatomical material is widespread over the United States and has been the subject of publicity in popular magazines[2] as well as professional journals.[3] Of the 39 states having medical and dental schools, less than a dozen provide for bequest of the body at death as an additional source of material for scientific study. Louisiana is one of these.[4] Thus far only several bequests have been received, but the experience of schools in California has shown that bequest is potentially a major source of supply. Physicians can perform a great service, in their contacts with patients and others, if they will discreetly pass on this information about a critical need and the way in which interested persons can contribute to human welfare.

An information sheet that has been prepared for distribution to inquirers is copied below (minus its headings), together with the form of bequest that has been approved by the Office of the Attorney General of Louisiana.

The Louisiana law provides that any resident of the State, of the age of 21 years or older and of sound mind, may bequeath his entire body at death or any part thereof for the purpose of advancing medical science. Persons who arrange for this disposition of their bodies have the satisfaction of knowing that the remains will be of lasting benefit in serving humanity. They have further satisfaction in relieving the survivors of the expenses attached to burial.

Bodies may be bequeathed to the Louisiana State Anatomical Board. This organization, operating under Louisiana law, is responsible for the procurement, use and final disposition of the human bodies that have been dedicated to scientific study. Such studies are carried out by three teaching institutions: Louisiana State University, School of Medicine; Loyola University of the South, School of Dentistry; Tulane University, School of Medicine. They are the member institutions composing the Board. If so desired, a body may be bequeathed specifically to any one of these institutions.

The procedure of bequest is simple. A form for this purpose will be supplied, and the form (two copies) needs only to be signed by the person and by two competent witnesses who are residents of the parish in which the bequest is made. The completed forms are to be mailed to the Secretary-Treasurer of the Board (address below), who will file one copy with the clerk of the district court. The person should make sure that those who are near him—the physician and family members—know about the bequest; they should be informed also on promptly reporting the death to the Board (to its Secretary-Treasurer or the Dean of any one of the three Schools named above). When the notice of death is received the Board will send an undertaker to secure the body.

Further information will be gladly supplied on request.

Secretary-Treasurer
Louisiana State Anatomical Board
Tulane University
New Orleans 18, Louisiana
Telephone: UNiversity 6-2741,
Extension 233 or 235

LOUISIANA STATE ANATOMICAL BOARD
New Orleans, La.

I hereby bequeath my body, after death, to the Louisiana State Anatomical Board for the purpose of advancing medical science. (If the person making this bequest desires that the study be carried out in a particular institution of the Board, the underlining of its name in the following list shall constitute a specific assignment to that institution: (1) Louisiana State University, (2) Loyola University of the South, (3) Tulane University.)

Witness *Signature*

Witness *Address*

Parish of residence

Parish in which this bequest was executed

Date

Mail (two copies) to:
Louisiana State Anatomical Board
Tulane University
New Orleans 18, Louisiana

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THE PROBLEM OF CARDIAC HANDICAPPED CHILDREN IN LOUISIANA

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NEW ORLEANS

Recently a greater significance attaches to the problem of heart disease in children. Since rheumatic fever and congenital heart disease account for approximately 95 per cent of heart disease in children, a handicapped child who requires long term care and supervision is often the result. Rheumatic fever continues to be the major medical cause of death up through adolescence, despite a progressive decline in incidence over the past fifty years. Congenital heart disease because of continued interest and improvement in methods of diagnosis probably has a trend to increase. Admittedly, these diseases form a relatively small part of the overall problem of heart disease, but they are of no less importance to the individual who may be afflicted; and, we now have methods of approaching the problems which they produce.

There have been no adequate surveys for the state from which the incidence of rheumatic fever or congenital heart disease can be determined, but surveys [1] conducted elsewhere indicate that approximately 1 per cent of school children (ca. 10 - 12,000 in Louisiana) are afflicted with rheumatic fever and rheumatic heart disease. Autopsy studies [2] at Charity Hospital of New Orleans suggest that the incidence of rheumatic involvement of the heart is no less in Louisiana than in other areas.

As reflected in Table 1, congenital heart disease occurs approximately one-third as commonly as clinically recognized rheumatic fever and its sequela.

SCOPE OF A PROGRAM FOR CARDIAC CHILDREN

A plan for treatment of cardiac handicapped children in Louisiana has been outlined through the cooperation of the Louisiana State Health Department and the Louisiana Heart Association. This plan emphasizes the role the practicing

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TABLE 1

AVERAGE ANNUAL ADMISSIONS RATE* (1950-55) (TWO MAJOR CHARITY HOSPITALS OF LOUISIANA)

	Rheumatic Fever†	Rheumatic Heart Disease		Congenital Heart Disease
		With activity	Without activity	
New Orleans	116	196	83	148
Shreveport	42	27	25	45

* Data necessarily included readmissions; consequently, rate of admissions is slightly greater than number of patients.

† Discharge diagnosis according to Standard Nomenclature of Diseases terminology.

physician would play in extending care to all children with, or potentially with heart disease. Two chief centers ideally located geographically, New Orleans and Shreveport, are being considered to offer highly specialized services which may be needed by the physician in the care of his patient. The plans immediately include an extension of the diagnostic, acute and convalescent needs and clinic services at Charity and the Crippled Children's Hospitals in New Orleans. A roster of resource personnel has already been partly formed and a certain amount of interest has been stimulated by the Louisiana Heart Association and State Health Department in individuals who can aid in the initiation of a more comprehensive program—for a really adequate program must go beyond the current practices.

A complete program should promote the development and integration of services and facilities for the state. A project so designed should (1) insure that children with cardiac problems receive adequate and continuing care, (2) remove the label of falsely diagnosed cases, (3) encourage the use of methods for prevention of rheumatic fever and bacterial endocarditis, (4) find other children with rheumatic fever and congenital heart disease and (5) offer educational facilities where needed.

The initiation of this program, however, requires the concerted effort not only of the Health Department, Heart Association and practicing physicians but also of the medical schools, state hospital system, nursing personnel, school system, and social, occupational and vocational rehabilitation services. Facilities for prolonged convalescent care, programs against further development and recurrences of rheumatic fever and bacterial endocarditis, and means for bacteriologic studies are integral parts of the program. Briefly summarized, an overall program requires hospital, clinic and field services. The State Health Department can offer certain facilities but these need to be supplemented, and in addition to cooperation among the various organizations, a coordinator with clearly defined responsibilities is necessary to both activate and coordinate the program on local and state levels.

NATURE AND EXTENT OF FINANCIAL SUPPORT FOR ACTIVITIES OF A CARDIAC PROGRAM

Another aspect of the problem to be considered is the financial one. Financial support for the care of cardiac children is on a restricted basis: needs are being met by strictly private or strictly charity sources, both of which fail to provide an outline of prolonged convalescence, adequate prophylaxis, or follow-up observations.

ADEQUACY OF PUBLIC AND PRIVATE FACILITIES

The services presently available are inadequate, but this inadequacy comes about largely through a lack of organization. Facilities for certain specialized aspects, such as cardiac catheterization, and particularly cardiac surgery, exist in very limited amounts but the over-all needs could be met easily through planning and cooperation since many of the facilities and services referred to above are available.

EDUCATIONAL PROGRAM

Informative programs for the general public are currently being sponsored by the Louisiana Heart Association and Health Departments through radio, television, newspapers and public forums. Probably more important are the scientific sessions on cardiac problems being presented to physicians by the medical schools and Heart Association. However, there is yet a need for extension of information to other professional groups which may be directly involved in the care of the cardiac child.

SUMMARY

The scope of a program for the care of the cardiac handicapped child necessarily entails all of the considerations discussed above. Because of the many varied problems which will be encountered in the development of the program, cooperation of professional, technical and social services is required. These services in turn need direction by an individual or a group familiar with needs of our local area. Fortunately, advances in therapy made during the past few years offer means of preventing rheumatic fever and correcting or improving certain congenital heart lesions. What we need now is interest and support to put the program into effect.

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VOMITING OF PREGNANCY

A recent report by the Council on Pharmacy and Chemistry emphasizes what is known and what is not known about this condition that affects as much as one third of pregnant women. The report points out that there is much difference of opinion as to the frequency and the importance of the condition from a therapeutic standpoint. Estimates are varied in the review made by the Council from the statement that it appeared in 80 per cent of all pregnancies to the opinion of one consultant, who said that less than 1 in a thousand obstetrical patients required hospital treatment for this condition. The opinion of a panel of four consultants experienced in this field was solicited by the Council. It was felt that the term "nausea and vomiting of pregnancy" while previously used in reference to the mild and severe forms should be restricted to the condition commonly observed during the first to fourteenth weeks of pregnancy, as characterized by some disturbance in appetite and reactions to food in a fairly large percentage of women, possibly up to one third of pregnant women. The panel felt that the term "hyperemesis gravidarum" or "pernicious vomiting of pregnancy" was applicable to a few patients who exhibited intractable vomiting and signs of disturbed nutritional status, such as alteration of electrolyte balance, 5 per cent or more weight loss, ketosis, and acetonuria, with ultimate neurological disturbances, liver damage, retinal hemorrhage, and renal damage. It was felt that this grave disorder was infrequent among pregnant women who had proper early prenatal care and protection from serious emotional difficulties.

In regard to the cause of the condition there was agreement on at least two points of etiological responsibility—pregnancy and psychological factors. The approach to the problem on the basis of an unusual hormonal stimulus and treatment with estrogen and progesterone has not been successful in alleviating the situation. The theory of a toxic substance produced during the first sixteen weeks has not been substantiated. It was considered that psychological factors played a major role in both mild and severe nausea and vomiting of pregnancy. However, there are occasional instances of severe vomiting occurring in the unexpected pregnancy of a previously sterile woman. In such instances the vomiting precedes the suspicion or substantiation of the diagnosis of pregnancy.

It was advised in all instances of nausea

and vomiting associated with pregnancy that a complete examination be carried out to eliminate possible causes other than pregnancy. It was thought that simple measures should suffice when the diagnosis was the more or less physiological nausea and vomiting of pregnancy. These would include rest, sedation, alleviation of nervous and emotional stresses, and the ingestion of frequent small meals high in carbohydrate. Because the mild forms of vomiting may rapidly develop into the intractable form suddenly and without warning these patients should be kept under observation particularly during the first sixteen weeks of pregnancy until the symptoms subside. Hyperemesis gravidarum, however, requires active support as a nutritional problem, which can best be served by hospitalization with proper attention to electrolyte balance, nutritional requirements, and necessary supportive measures gven parenterally.

Most of the drugs tested for these conditions when the investigation included blind and double blind studies had about the same level of effectiveness as the placebo. The groups of drugs were: (1) antihistaminics and the related antimotion sickness drugs; (2) anticholinergics; (3) vitamins; and (4) depressants. As there is a specific deficiency of pyridoxine (vitamin B_6) this drug was thought to be of value in the treatment of hyperemesis gravidarum. It was reported that the depressant, chlorpromazine, may have considerable value in the treatment of hyperemesis gravidarum. It was stated that blind studies have not been performed but reports were made of cessation of symptoms in hyperemesis gravidarum in one to five days. It was further advised, however, that such patients be watched carefully for evidence of blood and liver damage, and since serious side effects have been associated with chlorpromazine its use should be minimized in the milder form of nausea and vomiting of pregnancy.

Considering the fact that vomiting is one of the commonest symptoms associated with functional and with organic disease in man and in the animal kingdom, it is not surprising that vomiting of pregnancy remains as baffling a symptom to the modern physician as to generations of his predecessors. It is a fortunate circumstance that although the cause is unknown facilities for its control and possible relief are becoming more available.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

REPORT OF PRESIDENT

The year 1955 presented many problems for the officers and committees of the Louisiana State Medical Society, but on the whole, much has been accomplished.

The Society was represented at the meeting of the AMA in Atlantic City in June, 1955 by your President, Secretary-Treasurer and Chairman of the Council on Medical Service and Public Relations. Drs. P. H. Jones and J. Q. Graves represented the Society in the House of Delegates.

During the regular AMA session, the question of admitting osteopaths on the same basis as medical doctors was argued very strongly on the floor of the House of Delegates after almost slipping by us. Finally, it was voted not to accept them at this time.

You are reminded that the AMA is making a much stronger effort in regard to legislative matters than ever before and is forwarding information to us as quickly as possible.

An interim meeting in Boston was attended and sessions of the House of Delegates were followed.

The Conference of Medical Society Executives held in Chicago in January, 1956 was attended by Dr. Cole, who reported that excellent ideas were presented by very capable executives on various phases of medical society problems. Dr. Elmer Hess, President of the AMA in a letter to the office of the Secretary-Treasurer, stated that

he was pleased with the results of this meeting where many new ideas were presented.

The Regional Legislative Conference in Chicago in October was attended by Dr. C. J. Brown. A similar meeting where important legislative problems were considered was covered by Dr. Cole in Dallas in November.

Drs. M. C. Wiginton and J. P. Sanders attended the Rural Health Conference in Portland, Oregon. The benefits of this conference continue to be important, and its activities are increasingly helpful in the rural areas. Rural medical care should continue to improve under its guidance.

The American Medical Education Foundation met in Chicago on January 22, and Dr. Edgar Hull has reported on problems of interest to us and of aid to our medical schools.

Many meetings have been attended throughout the state. The Third District Meeting in Lafayette; Second District Meeting in Harahan; Founders Day Meeting in Alexandria; East Baton Rouge Parish Meeting in Baton Rouge; and Fourth District Meeting in Shreveport were all attended by your President and Secretary. Your Public Relations Counselors and Legislative Consultant appeared before most of these groups.

On March 1, your President, and Dr. Cole and Dr. W. Robyn Hardy attended a fine meeting of the Fifth District and Ouachita Parish Medical Societies, where Dr. Hess was guest of honor and spoke to the Rotary Club and District Medical Society meeting.

The Louisiana Health Council met in New Orleans. This Society was represented by your President and Secretary, who found these sessions interesting and productive of better relations for the State Society. This Society is a valuable aid to state health problems, and it seems necessary to encourage their work. Many rural areas throughout the state continue to need their help.

The Senate Finance Committee held hearings through most of March. Our ablest and most interested doctors from all over the nation testified before the committee on HR-7225. Each state society was requested to send a representative and Dr. P. H. Jones appeared on March 23. He feels that much truth was spoken by the medical representatives, but we fear that many of our legislative representatives in Washington have already decided in favor of this bill.

Telegrams and letters were sent to our senators and members of the Senate Finance Committee, urging them to consider carefully two dangerous portions of this bill. Namely, total disability payment at age 50, and to lower age limit of pensions to women to age 62 instead of 65.

Malpractice suits continue to increase throughout the state with amounts awarded reaching astounding proportions. Our attorney, Mr. St. Clair Adams, has handled many cases for the Society and prosecuted many offenders for the State Board of Medical Examiners. All of us are cautioned to observe certain rules on prevention of malpractice suits, by avoiding unwise comment on questionable cases and by keeping complete records on all cases. Proper use of our Mediation Committee, with reasonable discussion with patients concerning fees, will materially reduce these harassing suits.

Questionnaires concerning malpractice insurance sent to our membership netted 957 replies. The Executive Committee felt that nothing further should be done at this time toward securing information from doctors about such insurance since the response to the two questionnaires sent out seemed wholly inadequate.

After many meetings, the special committee appointed by your President to select an Assistant to the Secretary recommended the employment of a Legislative Consultant and a firm of Public Relations Counselors to work with the Secretary-Treasurer's office. The Executive Committee approved the employment of these men. Both the Legislative Consultant and the Public Relations Counselors have been busy with their new work. The employment of the Public Relations Counselors and Legislative Consultant marks an important step forward in the proper management of our medical problems. A public relations program is under way and a code of ethics with television, radio and press is in operation. Our Legislative Consultant has been busy contacting doctors and legislators all over the state, trying to improve our position in political matters, particularly chiropractic. Both groups have appeared before parish and district societies frequently. The Executive Committee is to be congratulated on the approval of the employment of the Public Relations Counselors and Legislative Consultant.

The Council on Medical Service and Public Relations and Dr. Cole are also to be congratulated on the monthly newsletter "Capsules", giving current information of interest to the medical profession. You are urgently requested to read this letter carefully, as a great amount of time and effort is expended in getting this bulletin out to you.

Much time has been spent by your officers and many members of the Society in education of the public and working against Chiropractic. The Committee on Public Policy and Legislation, Dr. Boyd, Chairman, and our Legislative Consultant have worked hard on this problem which comes up every legislative year. Strong efforts are still necessary to post our membership and educate the public about this menace.

Many special and standing committees have done much work, and I want to express my appreciation for their work. Some of these are: Public Policy and Legislation Committee, Dr. Elmo Boyd, Chairman; Committee on Congressional Matters, Dr. C. J. Brown, Chairman; Committee on Child Health, Dr. Sims Chapman, Chair-

man; Committee on History of Medicine in Louisiana, Dr. Isidore Cohn, Chairman; Committee on Federal Medical Services, Dr. I. Gajan, Chairman; Council on Medical Service and Public Relations, Dr. Robyn Hardy, Chairman.

I desire to express my sincere thanks for the privilege of serving as President to the Society as a whole, to all the committees, and especially to the Executive Committee for their cooperation during 1955-56. I want to thank, particularly for their counsel and support, Dr. C. Grenes Cole, Secretary-Treasurer, who has been especially helpful to me with sound advice, Drs. Robyn Hardy, Ashton Thomas, and P. H. Jones. Recognition and thanks are also given to Miss Annie Mae Shoemaker, Assistant Secretary-Treasurer, and the office personnel for their cheerful and efficient work.

Recommendations

1. Questionnaire to secretaries of parish medical societies and councilor of each district, in regard to the activities of respective societies.
2. Meetings of presidents and secretaries of the parish and district societies to be held semiannually, to better coordinate efforts for improvement of Organized Medicine.
3. District councilors make every effort to better organize the district and parish societies.
4. Continue membership in LOSL and the committee representing the State Society be selected from the personnel of the Committee on Public Policy and Legislation.

MAX M. GREEN, M. D., President

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MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

PHYSICIANS REPORT ON USE OF NEW SYNTHETIC HORMONE

A preliminary report on the use of a new synthetic hormone to make up for hormone gland activity lost through disease or surgery was made recently by two Ohio physicians.

The hormone, fludrocortisone acetate, appears to be 15 to 20 times as effective as hydrocortisone, another synthetic now used for such conditions.

The greatest usefulness of fludrocortisone probably will be in adrenal insufficiency (a disorder in which the adrenal glands fail to function properly) and in cases of surgical removal of the glands in cancer or other serious illnesses, they said.

The physicians, who made their report in the Dec. 24 Journal of the American Medical Association, gave the hormone to patients with a variety of disorders, including a serious nervous condition characterized by loss of appetite, and eye disease, rheumatoid arthritis, and adrenal cortical hypofunction.

The hormone is similar to cortisone and hydrocortisone in its ability to inhibit the pituitary stimulation of the adrenal gland; produce loss of nitrogen, calcium, and phosphorus; inhibit inflammation, and produce a sense of well being.

It also resembles desoxycorticosterone, another synthetic hormone, in its effect on salt, water, and potassium balance. However, its use may be limited in conditions such as rheumatoid arthritis in which it causes too much salt and water retention, they said.

The report was made by Drs. George J. Hamwi and Robert F. Goldberg, from the division of endocrinology and metabolism, department of medicine, Ohio State University, Columbus, Ohio.

NEW DRUG FOR MENTALLY ILL

New aid for the mentally ill was promised by Dr. Nina Toll in a paper presented before the

Section on Medical Sciences, American Association for the Advancement of Science meeting in Atlanta, December 27.

A new drug helps make office psychotherapy wth schizophrenic patients more effective and shortens the course of therapy, Dr. Toll reported. Patients taking the drug are better able to understand their symptoms, can talk about their illness, and become more alert. As a result, therapy is more effective and the illness shorter in many cases.

Frenquel is the drug which proved so helpful. It is the new anti-hallucinatory, anti-confusion drug discovered in the research laboratories of The Wm. S. Merrell Company, Cincinnati drug firm.

COMMITTEE OUTLINES PROGRAM FOR POISON CONTROL

The American Medical Association's Committee on Toxicology has outlined four methods for combating the perennial problem of accidental childhood poisonings.

The methods include education, more stringent laws, establishment of poison centers, and greater efforts by local physicians. They were discussed in a report prepared for the committee by Dr. Jay M. Arena, Durham, N. C., and published in the Dec 17 Journal of the American Medical Association.

Bernard E. Conley, Secretary of the committee, said " . . . the curiosity of the children coupled with the casualness with which many parents handle and store drugs and chemicals are predisposing factors to most unintentional poisonings."

The "alarming feature" of the problem is the regularity with which various household agents and drugs are swallowed by children, the report said. Leading causes are drugs, especially aspirin and barbiturates, petroleum products, lead, corrosive agents such as lye, and arsenic.

ARE YOUR DOCTOR'S BILLS TOO HIGH?

Who in the world doesn't grumble about a doctor's bill now and then? Are the "men in white" justified in charging the fees they do; or are they gigging the helpless public?

These questions and other provocative ones in their connection are answered in an illuminating article on "The Cost of Educating a Doctor" in the February issue of Know the FACTS magazine published in Washington, D. C. by Oliver Presbrey, with Martha Rountree as editor-in-chief.

Specifically, the article stresses the high cost, in time and money, required of medical aspirants. A person who wants to be a doctor can expect to pay as much as $37,000 in the course of fourteen years he will spend preparing to hang out his shingle.

Yet this almost prohibitive expense in time and money has not been a deterrent to persons wanting a medical career. "There are 210,886 doctors in the U. S. today with 28,254 men and women studying medicine in 75 medical institutions," states this discussion. What is the reward? "A chance to make more than $28,000 annually (after several years of practice); a chance to make medical history (research); and an opportunity to render invaluable service to mankind and the community."

PEDIATRIC COURSES TO BE CONDUCTED AT THE CHILDREN'S HOSPITAL OF PHILADELPHIA

The following short courses will be conducted at The Children's Hosipital of Philadelphia in May and June 1956.

1. *Pediatric Advances for Pediatricians and General Practitioners.* May 28 through June 1, 1956. A Refresher Course conducted by the Staff of the Children's Hospital of Philadelphia, in collaboration with the Department of Pediatrics of the University of Pennsylvania and the Camden Municipal Hospital. Tuition—$100.00.

2. *Practical Pediatric Hematology.* June 4, 5 and 6. Conducted by Dr. Irving J. Wolman and other members of the Hematology Department of the Children's Hospital, under the auspices of the Graduate School of Medicine, University of Pennsylvania. Tuition—$60.00.

3. *Blood Group Incompatibilities and Erythroblastosis Fetalis.* June 7 and 8. Conducted by Dr. Thomas R. Boggs, Jr. of the Philadelphia Serum Exchange of the Children's Hospital of Philadelphia, under the auspices of the Graduate School of Medicine, University of Pennsylvania. Tuition—$50.00.

For information write to Irving J. Wolman, M. D., Children's Hospital of Philadelphia, 1740 Bainbridge Street, Philadelphia 46.

LOUISIANA HEART ASSOCIATION

The second scientific session and first general assembly meeting of the Louisiana Heart Association, an affiliate of American Heart, will be held at the Tulane University auditorium on May 19 with registration scheduled for 9 a.m., Dr. Frank W. Pickell of Baton Rouge, president of the Association, announced recently.

Principal speaker will be Dr. Albert Dorfman, Associate Professor, Department of Pediatrics, Bobs Roberts Hospital, University of Chicago,

Also scheduled to speak are Dr. Charles A. Beskin of Baton Rouge and Dr. George Wilson of Shreveport.

TRANQUILIZING DRUGS

A "new ray of hope," the tranquilizing drugs; plus more psychiatrists, offer the best answer to the rising incidence of mental illness. Dr. Nicholas A. Bercel of the University of Southern California School of Medicine told a meeting of the

American Pharamaceutical Manufacturers Association in Los Angeles.

He discussed three of the more widely used tranquilizers, reserpine, chlorpromazine and meprobamate and reported that meprobamate is the "best, safest and simplest drug for ordinary tension that besets so many otherwise healthy people." He also suggested the possibility of its use by the average "nervous" person to eliminate the use of alcohol and barbiturates.

The anatomy of anxiety and the inadequacies of earlier drugs and psychotherapy in eliminating the symptom were outlined. Barbiturates and other sedatives were held inadequate because of their action on general rather than specific areas of the nervous system and because of the problems of tolerance, addiction and impermanence of action.

NEW PSYCHIC STIMULANT

Ciba Pharmaceutical Products recently announced the introduction of a new stimulant and anti-depressant called Ritalin.

The new product, which has been tested clinically on more than 2,000 psychiatric and non-psychiatric patients during the past 15 months, has been available for prescription since March 1st.

Ritalin (methyl-phenidylacetate hydrochloride CIBA) is described by Ciba as a "mild cortical stimulant which counteracts oversedation, lassitude, depression and certain other side effects of barbiturates, antihistamines, chlorpromazine and rauwolfia derivatives."

Because no appreciable increase in blood pressure and pulse rate has been recorded in the use of Ritalin, it has also proved valuable in the treatment of patients with hypertension who have been previously treated by tranquillizing drugs alone.

GALACTOSEMIA

The cause of an often fatal metabolic disease of children has been discovered by scientists of the Public Health Service's National Institute of Arthritis and Metabolic Diseases, Surgeon General Leonard A. Scheele announced on March 12.

Drs. Herman M. Kalckar, Elizabeth P. Anderson, and Kurt J. Isselbacher, in work conducted at the National Institutes of Health, Bethesda, Maryland, have unraveled much of the mystery surrounding the little understood children's disease, galactosemia, also known as galactose diabetes.

This disease ordinarily appears within a few days after birth. The infant suffering from galactosemia is unable to utilize or even tolerate milk in any form. Lactose, often called milk sugar, contains another sugar, galactose. This substance cannot be handled by the child's system if he has galactosemia.

The Institute scientists have discovered a hitherto unknown enzyme in normal red blood cells, which they call P-Gal transferase. This enzyme, they found, is necessary to complete conversion in the body of galactose into glucose, the common sugar of the blood.

STUDY SHOWS MOST EFFECTIVE SEASICKNESS DRUGS

Chances of seasickness are lowest among passengers who are under 25, slightly thin, traveling midship on a second or third voyage—and are supplied with one of 11 drugs recently proved to be helpful.

A five-month study of 26 anti-motion sickness drugs among almost 17,000 servicemen showed that 11 of them were helpful and three of these were more effective than the rest.

The study was reported in the March 3 Journal of the American Medical Association by an Army, Navy, and Air Force motion sickness team.

The tests were run from Nov. 15, 1954, to April 10, 1955, on military transports crossing the Atlantic. Ten eastward and five westward crossings were used. Passengers were given unmarked pills which were either drugs or placebos. Placebos are inactive substances used as a yardstick against which the action of real medicines can be measured.

The study showed that susceptibility to seasickness decreased with age; that individuals quartered fore and aft were more susceptible than those in midship; that heavier individuals were slightly, but not significantly, more susceptible than their lighter colleagues. The activities of the men while on board had no relation to their susceptibility to illness.

Motion sickness was twice as frequent in persons who had had it before as in those with no history of sickness. However, seasickness was less frequent on westward trips—possibly because all the passengers had had at least one previous crossing.

The top three drugs by trade name are Bonamine, Phenergan, and Marezine when given three times daily. All showed prolonged activity, the team reported.

"STINGLESS" FIRST AIR CREAM ANTISEPTIC INTRODUCED BY JOHNSON & JOHNSON

A new cream-type medication that provides antiseptic action without stinging or pain in treatment of cuts, abrasions, burns, chapping, sunburn and insect bites has been developed by Johnson & Johnson, it was announced recently.

Known as First Aid Cream, this latest addition to the Johnson & Johnson product line is the result of seven years of intensive research to develop a complete medication. First Aid Cream fights infection, relieves pain and aids healing simultaneously. It can be applied directly to open wounds and will not cause stinging or pain

that usually results when old-fashioned harsh antiseptics are used.

Following a series of exhaustive tests in clinics, hospitals and households across the nation, First Aid Cream now is being made available to the general public. It is being marketed through the usual retail outlets which handle Johnson & Johnson products.

"BATH LIFT" FOR INVALIDS

With a newly-devloped apparatus, the infirm and those incapacitated by paralysis, rheumatism and similar disorders can get into or out of a bath unaided, and without difficulty or danger, say the British firm who designed and manufactured it. Successful trials were carried out at Stoke Mandeville Hospital. The "bath lift" has a seat with backrest which is placed in the water and raised to the rim of the bath by water pressure. The patient sits on the seat, lifts his legs into the bath and, on pushing a lever, is lowered gently into the water. A contrary movement of the lever returns the seat to its original position. A rubber tube from the tap to the base of the apparatus supplies the hydraulic pressure (normal mains pressure of 15 lb. per sq. in. will raise a weight of 490 lb.).

(Levit Engineering Co., Summer Road, Croydon, Surrey, England.)

DR. TABB TO SPEAK AT AMA CONVENTION

Dr. Harold G. Tabb, of New Orleans, Louisiana, will discuss the subject of *Fractures of the Facial, Orbital, Maxillary and Their Management*, at the American Medical Association Convention in Chicago, Illinois to be held June 12-14.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

The thirty-third annual convention of the Woman's Auxiliary to the American Medical Association will be held in Chicago at the Conrad Hilton Hotel, June 11 through June 15, 1956. Convention Chairman is Mrs. Leonard J. Houda and Co-chairman, Mrs. Maurice M. Hoeltgen.

A cordial invitation is extended to all members of the Woman's Auxiliary, their guests and the guests of physicians attending the convention of the American Medical Association, to participate in all social functions and attend the general meetings of the Auxiliary.

Headquarters will be at the Conrad Hilton Hotel. Tickets for the various social functions will be available at the registration desk only. The Hospitality Room will be in the Normandie Lounge.

It is requested that members and guests register early and obtain their badge and program. Registration will begin at 12:00 noon on Sunday.

One of the highlights of the meeting will be a luncheon on June 12, in honor of the Past Presidents of the Woman's Auxiliary at which Mrs. Mason G. Lawson, President, will preside. Guest Speaker for this occasion will be Mr. Leonard E. Read, President of the Foundation for Economic Education, who will speak on "The Positive Approach to Combatting Socialism."

BOOK REVIEWS

Pomp and Pestilence. Infectious Disease, Its Origins and Conquest; by Ronald Hare, Professor of Bacteriology in the University of London. New York, N. Y. Philosophical Library, Inc., 1955. pp. 224, $5.75.

This neat pocket-sized volume, lacking illustrations, provides a comprehensive account of the progress made in the elucidation of the epidemiology of infectious diseases and the application of effective measures for their control. The story is told so simply that at first glance the information may seem trivial, but on more careful reading it is discovered that the author has the rare facility of presenting technical data in terms which an intelligent layman can appreciate.

All infectious agents are defined as "parasites", irrespective of their etiologic classification or their host-parasite relationship. It is heartening to have a microbiologist discuss animal parasites with the same understanding as he does bacteria and viruses. Moreover, with equal familiarity the author considers the effects of pathogens on the individual and on the human community.

The format of the book is conservative but attractive; the text is free of typographic errors, well documented and there is a useful combined author and subject index. Because of its accuracy and broad comprehension of the problems discussed, the volume should provide the physician with profitable leisure reading. And he can recommend it to those of his patients who desire authoritative information on infectious diseases.

ERNEST CARROLL FAUST, PH.D.

Hemorrhage of Late Pregnancy; by John S. Fish, M. D., Springfield, Ill., Charles C Thomas, 1955. Pp 180, Price $5.50.

In a most comprehensive review of the subject, the author discusses placental anatomy, clinical characteristics and diagnosis, as well as placental diagnosis of hemorrhage in late pregnancy. Of 5,806 private cases, the incidence of late pregnancy hemorrhage by cause was found to be as follows: rupture of marginal sinus 39 per cent, placenta previa 30 per cent, abruptio placentas 10 per cent, and undetermined 10 per cent.

Fish and his associates in the Bartholomew group have shown that bleeding from rupture of a marginal sinus in the placenta is more common than generally accepted. The illustrations are excellent and the entire book is informative. It would be easier reading if there were not numerous instances of sentences eight to twelve lines long.

EUGENE H. COUNTISS, M. D.

Cerebral Vascular Diseases; Transactions of a Conference Held Under the Auspices of The American Heart Association, Princeton, New Jersey, January 24-26, 1954; Irving S. Wright, Chairman, E. Hugh Lucky, Editor, New York, N. Y. Grune & Stratton, 1955, Pp 167, Price $5.50.

This is a series of fourteen presentations and discussions by 34 conference members, on cerebral vascular disorders. In addition to the conventional topics of anatomy, physiology, pathology, diagnosis and treatment, there are presentations dealing with the emotional problems of cerebral vascular disease and their sociologic, economic and personal significance.

The volume provides a good survey of the present status of this field. Dr. H. Houston Merritt's review of neurologic diagnostic aspects shows very little change since his well known treatment of the same subject that is now about twenty years old. This reviewer regrets that more time and space were not allowed to Drs. Adams, Scheinker and Zimmerman to elaborate their respective ideas concerning the pathology of cerebral vascular diseases. It is interesting to see the extent to which careful workers in this basic field may still disagree about the mechanisms underlying macroscopic changes seen in apoplexy. The chapter by Dr. Rusk on rehabilitation deserves the widest diffusion because of its immediate usefulness in increasing the salvage rate among hemiplegics. His opportunities for evaluating rehabilitation measures are perhaps unique, and his presentation should help to dispel the atmosphere of therapeutic gloom which has traditionally surrounded the hemiplegic patient.

Presumably to reduce the cost of the volume, the slides used by the speakers in the original conference were not reproduced. One occasionally misses their presence. Some of the presentations have an adequate bibliography appended; several have none, and this was sometimes regrettable, notably in the chapters dealing with pathology and the physiology of the cerebral circulation. Should not one of the purposes of this volume be to whet the appetite of the busy nonexpert?

E. B. FERGUSON, JR., M. D.

Animal Agents and Vectors of Human Disease; by Ernest Carroll Faust, Ph.D., Philadelphia, Lea and Febiger, 1955, pp. 660, with 216 text figures and 9 plates, Price $9.75.

This new text book of Parasitology by Professor Faust covers about the same range of subject matter as Craig and Faust's "Clinical Parasitology". It is somewhat briefer, particularly in the clinical aspect of the infections, and the subject matter is differently arranged so as to place the natural history, pathological and clinical manifestations, diagnosis, treatment and prevention of all parasitic infections together in preliminary chapters. This has value in tabular presentation, and shortens the text dealing with individual parasites, but requires search for data and leads to an occasional omission.

The data on each parasite are presented in Dr. Faust's concise, authoritative and comprehensive manner, and give the latest information available. Most of the excellent illustrations are Dr. Faust's own drawings, many taken from his previous publications. The only unfortunate economy is in the plates of malaria parasites which are in black and white instead of color. The section on Technical Aids includes a variety of approved methods of examination in accurate detail. The list of references to the literature at the end of each chapter includes most of the important sources. Other references in the text would be of more value to the reader if a brief indication of the publication were included.

This book will undoubtedly become widely used as a textbook for students of medicine and parasitic biology, and should be in the library of every individual and institution interested in the animal parasites of man. The author and publishers are to be commended on an excellent product.

HENRY E. MELENEY, M. D.

Psychocutaneous Medicine; by Maximilian E. Obermayer, M. D., Springfield, Illinois, Charles C Thomas, 1955, Pp 487, Price $9.75.

Whether, and to what degree, emotional factors are responsible for many of the common dermatoses, is moot. Dr. Obermayer believes that psychosomatic factors are of great importance in skin diseases and collects much evidence in support of his belief. Yet he makes a real effort to preserve an unbiased viewpoint; contrary views are also incorporated into his book. This reviewer found the section on stigmatization most interesting. The book should have a wide appeal.

VINCENT M. DERBES, M. D.

The Therapy of Skin Tuberculosis; by Gustav Riehl and Oswald Köpf, Springfield, Illinois, Charles C Thomas, 1955, Pp. 247, Price $6.75.

Though skin tuberculosis is not a common disease in this country cases continue to be seen. Thus we are fortunate to have a very thorough and scholarly book on the subject by a man with extensive experience with this condition. Professor Riehl, after whom Riehl's melanosis takes its name, has done an excellent job.

VINCENT J. DERBES, M. D.

Reflections on Renal Function; by James R. Robinson, M. D., Springfield, Charles C Thomas, 1954, pp 162, Price $3.50.

This is an interesting and refreshing small monograph based on lectures given to students in Cambridge. As the author states, it is intended to bridge the gap between the chapters in general textbooks and larger treatises such as that of Homer Smith. The author contends that "renal physiology is controversial and perhaps ought to be more so. There is a temptation, especially when teaching, to seek clarity by a too well organized presentation which gives the impression of a greater understanding than we really have." The author has steered a middle course, but, in doing so, has indicated his impressions of the various issues and problems in a clear, informal, and critical manner.

The presentation of clearance concepts is excellent, as is the discussion of glomerular filtration and tubular reabsorption and secretion. Glomerular filtration is conceived as a variable and the possibility presented that protein is filtered in small amounts by the healthy glomerulus and subsequently reabsorbed by the tubule. It is suggested that the main function of the kidney is to safeguard the composition of extracellular fluids by conserving soluble constituents of the body. The tubular epithelium thus becomes a most important gland of internal secretion, for it secretes no less than 180 liters of extracellular fluid into the blood every day. This implies that a diuretic which increases the urine output without increasing glomerular filtration rate should be classed as an inhibitor and not a stimulant of renal activity. The suggestion is also made and evidence cited that the loops of Henle may have the function of assisting the bulk-reduction function of the proximal tubules by passive reabsorption of water and salts, thus allowing the attainment of osmotic equilibrium between the fluid passing through the thin limb and the blood in the vasarecta. The "counter-current" hypothesis of Hargitay and Kuhn for the production of a hypertonic urine is discussed in detail in connection with this function of the loops of Henle and the question raised as to its significance in man. The discussion of the mechanism of action of the anti-diuretic hormone is thought-provoking. By analogy with results on frog skin, the action of the hormone may be that of opening up of pores and the reabsorption of osmotically free water in the distal tubule.

The above examples should serve to illustrate the nature of these "Reflections." Throughout the discussions of the excretion of water and sodium, renal control of acid-base balance, regulation of renal function, regulation of body fluids, there is a delightfully told story, combining the historic and traditional points of view with the suggestions from recent work. The problems are summarized in an epilogue which might well be read before as well as after the rest of the material. The monograph is heartily recommended to students and physicians as a stimulating, provocative and timely account of what physiologists believe to be the mechanisms by which the kidney performs its valuable functions.

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PUBLICATIONS RECEIVED

Little, Brown & Co., Boston: Therapy of Fungus Diseases, edited by Thomas H. Sternberg, M. D., and Victor D. Newcomer, M. D.

Prentice-Hall, Inc., Englewood Cliffs, N. J.: The Truth About Cancer, by Charles S. Cameron, M. D.

W. B. Saunders Co., Phila.: The Neuroses in Clinical Practice, by Henry P. Laughlin, M. D.; Clinical Recognition and Management of Disturbances of Body Fluids, by John H. Bland, M. D. (2nd edit.); Electrocardiography, Fundamentals and Clinical Application, by Louis Wolff, M. D., (2nd edit.).